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MANUAL

OF

PLANT DISEASES

BY

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PREFACE TO THE SECOND EDITION

No other excuse need be offered for a second edition than the rapid output of plant-disease literature during the six years that have elapsed since the first edition. An attempt has been made to incorporate the new researches, especially those which bear directly upon the types selected for detailed consideration. In order to conserve space for the additions, it has seemed necessary to omit the detailed treatment of a few types given prominence in the first edition. Many parts of chapters have been entirely rewritten, but the limitation of space has made it impossible to include many features which might seem desirable. In the lists of important diseases at the end of most of the chapters, only types are selected, with key references which should serve as a starting point if the student wishes to make a special study of a given disease. New key references have been added and some of the older ones omitted.

The author wishes here to express his gratitude for the words of appreciation and the friendly advice and criticisms which have been received from various plant pathologists in this country and abroad since the appearance of the first edition. Special acknowledgment is due to my coworkers in the Department of Plant Pathology, Dr. L. K. Jones, Dr. G. A. Huber, Dr. C. S. Holton, Mr. Kenneth Baker and Dr. Grover Burnett, for suggestions and aid, and especially to the last named for assistance in preparing the index and also for a very complete compilation of the literature on virus diseases which has been drawn upon in the revision of Sec. III.

F. D. HEALD.

STATE COLLEGE OF WASHINGTON,
PULLMAN, WASH.,
December, 1932.

PREFACE TO THE FIRST EDITION

Fifteen years ago the plant pathologists of America welcomed the appearance of "Fungous Diseases of Plants," by B. M. Duggar, as the first general text or reference book of American origin to occupy the field. Although a number of books have since been written covering special phases of plant pathology, no general manual has appeared to serve as a guide in classes in general plant pathology, which has now become an accepted part of the agricultural curriculum. The pressing need for a modernized book for use in the author's classes was the motive that prompted the preparation of this book. The plan of presentation of the subject is essentially that which has been followed in the author's classes for the last 10 years.

An attempt has been made to present a view of the whole field of plant pathology, including environmental and virus diseases as well as those of bacterial and fungous origin, as it is felt that a book of restricted scope would perpetuate an erroneous notion which has been prevalent in recent years as to the real province of plant pathology. It has also seemed advisable to avoid the dictionary type of presentation and to give instead a detailed consideration of a much smaller number of diseases, with the hope that this method of approach will impress the student with the importance of the subject and stimulate detailed rather than superficial study. Additional types with a few guiding references are given with the expectation that the student may be led to develop these according to the general plan.

No attempt has been made to present an organized treatment of culture methods and general plant pathological technique or of the principles and practice of disease control, since, with the rapid strides made in recent years, it is the practice to present these phases of the subject in separate courses. Special manuals covering these subjects would be welcomed by all teachers. In the treatment of the parasitic diseases, it has seemed desirable to follow the taxonomic sequence rather than host groups, since it is not generally possible to require systematic mycology as a prerequisite to the elementary course in plant pathology. It has been the aim to introduce enough systematic mycology to provide for this lack of mycological preparation. The order of presentation may not suit all teachers. In actual practice, it may be varied if desired by passing to Sec. IV, Parasitic Diseases, immediately following the two introductory chapters and then returning to a consideration of the non-parasitic and virus diseases.

A number of principles have guided the selection of the diseases given detailed treatment. Consideration has been given to types of disease, economic importance, causal factors or pathogenes and control methods. Limitation of space is the only excuse for the omission of certain important diseases which would otherwise have been introduced. It is felt that the historical introductions are well worth attention, as they will serve to impress the student with the fact that our present knowledge rests upon a long series of painstaking researches but that in most cases the field is still open for new discoveries.

The author is indebted to a large number of pathologists for illustrations, which are credited in the legends, and to other pathologists for suggestions during the progress of the work. Special acknowledgment is here made to the late Dr. C. V. Piper for suggestions and criticisms covering the entire manuscript and to the following for a critical reading of portions of the manuscript: Dr. L. R. Jones, Dr. I. E. Melhus, Dr C. T. Gregory, Dr. F. A. Weiss, Dr. Fred R. Jones, Prof. J. B. S. Norton, Dr. G. W. Keitt, Prof. H. P. Barss, Dr. L. K. Jones, Mr. D. F. Fisher, Dr. A. J. Mix, Prof. F. C. Reimer, Dr. A. J. Riker, Dr. W. H. Martin, Dr. C. W. Hungerford, Dr. Wanda Weniger, Dr. N. J. Giddings, Dr. L. W. Durrell, Dr. M. F. Barrus, Dr. L. R. Hesler, Prof. R. E. Smith, Prof. L. E. Melchers, Dr. J. J. Taubenhaus, Dr. Haven Metcalf, Mr. G. F. Garvatt, Mr. R. P. Marshall, Dr. E. C. Stakman, Dr. V. F. Tapke, Dr. N. A. Cobb, Dr. William Trelease, Dr. G. L. Peltier, Dr. H. T. Güssow, Prof. W. T. Horne, Dr. Charles Brooks and Prof. F. C. Stewart. In general, the plan was followed of submitting portions of the manuscript for critical reading to those workers who had previously made a special study of the subjects covered. The author has incorporated many valuable suggestions received from these sources and here wishes to express his appreciation for the assistance rendered. For the final form of the manuscript, the author alone is responsible.

The author is also indebted to his coworkers Prof. B. F. Dana, Mr. E. E. Honey and Mr. G. L. Zundel for suggestions and aid during the progress of the work.

F. D. Heald.

State College of Washington,
Pullman, Wash.,
September, 1926.

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CHAPTER I

INTRODUCTION

Plant pathology is that phase of botanical science which deals with the *diseases* or *troubles* of plants. The knowledge concerning plant diseases now constitutes the field of *phytopathology* (Greek: *phyton*, plant; *pathos*, disease; *logos*, discourse), a science which has come to rank with horticulture, agronomy and soil science in the realm of plant industry. The *phytopathologists* are the trained plant doctors, the "medicine men of agriculture," whose final goal is successfully to prevent or control plant or crop diseases.

Disease Defined.—It is important that a clear concept of disease, as applied to plants, should be gained at the very outset. In discussing disease, we may consider plants from two different points of view: first, as individuals whose place in nature is filled by growth to maturity with provision for the reproduction of their kind; and, second, as producers of crops, economic products or possessors of qualities desired by man. The first aspect of disease may be called the individual aspect, while the second may be designated as the agricultural or commercial aspect.

First, disease in plants may be defined as any variation from the normal, as expressed either by the checking or by the interruption of physiological activities or by structural changes, which are sufficiently permanent to check development, cause abnormal formations or lead to premature death of a part of the plant or of the entire individual. Diseases may be localized, affecting only special organs or parts, such as roots, stems, leaves, flowers or flower parts or fruits, or only certain portions of these organs may be involved. *Localized diseases* stand in marked contrast to those which pervade or affect the entire plant and may, there-

fore, be classed as *systemic* troubles. A few illustrations will suffice to make this concept of disease clear. If a plant shows minute or more extensive yellow spots or dead areas upon its foliage, indicating impairment or complete and permanent interruption of the chlorophyll function of localized areas, the photosynthetic activity or the food-manufacturing power of the plant is impaired or lessened just in proportion to the amount of green tissue that is put out of business. Such a plant is as truly diseased as one which has suffered a derangement that has caused its entire foliage to become yellow or devoid of green pigment. The roots of a plant may suffer changes which retard or reduce the absorption of water or prevent absorption entirely, and the plant may suffer from water shortage or it may wither and die as a result of these local disturbances. Whether these disturbances are slight or profound, disease results.

Localized killing of bark and cambium at any point on trunk or branches, if the killed area completely girdles or encircles the structure, will result in the death of all parts distal to the lesion; and if the lesion is on the main axis, the entire plant may be killed, as the root system will be starved, due to the breaking of the path along which the elaborated food travels from the foliage to the underground structures. Flowers may be blighted or fruits rotted by local disturbances which have no effect upon the physiological processes of other organs, or these changes may come as the result of rather deep-seated disturbances in the nutrition of the entire plant body. All plant parts are likely to suffer pathological changes, and there are no physiological processes that are free from possible derangement. The physiological processes of absorption and conduction of water and solutes; carbohydrate and proteid synthesis; digestion, respiration and complicated metabolic processes; translocation of elaborated food; transpiration; growth; and reproduction in a healthy or normal plant are in delicately adjusted balance the equilibrium of which may be upset by the operation of either internal or external factors.

Second, disease may be defined as a "failure of thrifty development or failure of the plant to produce a commercial product of satisfactory quality and quantity" (Smith, R. E. and Smith, E. H, 1911). The first portion of this definition alone, "failure of thrifty development," would not suffice for our agricultural or commercial concept of disease, since there are various cases in which a plant makes an especially thrifty or vigorous vegetative development but yields a product of poor or undesirable qualities. For example, apple trees which are apparently the picture of health may yield a crop of fruit seriously damaged by Baldwin spot or bitter pit; or wheat which has all the external appearance of vegetative vigor may yield an abundance of "yellow berry" or starchy kernels. The commercial product may suffer in quality only, or in many cases there will be a lowering of quality or grade and a reduction in quantity. Fruits may be disfigured or deformed, and their keeping

qualities lowered; tubers and root crops may exhibit external or internal defects; cereals may produce shriveled or shrunken grains, or these may be blackened with smut; hay or forage crops may be lowered in nutritive properties, and forest products may be stained or partially disintegrated. In all these cases, quality has been lowered as a result of the diseases responsible for these defects. As a general thing, severe foliage diseases are likely to cause a reduction in yield, since yield is so intimately related to the carbohydrate manufacture; fruits may be fewer and smaller, tubers fewer and smaller, roots reduced in size and forage crops lowered in tonnage per acre.

Agricultural or commercial demands have established certain desirable characteristics which should be fulfilled by "crop" or cultivated plants. Some of these qualities, which are desirable from the commercial standpoint, may be detrimental to the thrift or health of the plant. The variegated or golden varieties of our ornamental plants, prized for their decorative value, would be classed as sick plants according to our first definition. Could we say that the wild parsnip with its slender root is less healthy than its cultivated neighbor with its root gorged with reserve food? Many conditions which are prized in cultivated plants are not the necessary accompaniments of a healthy or thrifty development. The above concepts of disease open a wide field for the plant pathologist and reveal the close relationship between his province and those of the agronomist, horticulturist and soil chemist or physicist.

Needs or Requirements for a Thrifty Development.—Since thrifty development has been emphasized in our concept of health, it is proper that the needs or requirements for a thrifty development should be briefly enumerated:

1. The proper inherent qualities of seed or stock from which plants are to be grown. This may be expressed in another way by saying that the parents of our crop plants should be selected. Seed or stock apparently sound or free from disease may really be carrying disease in a latent form. In such cases, disease may appear in the crop, even though climatic and soil factors offer everything that is desirable.

2. The proper environmental conditions of both air and soil—proper moisture, temperature and light relations; proper physical composition and aeration of the soil; and proper chemical composition of both soil and air in order that food materials may be available and toxic or poisonous substances be absent. For any given crop plant or variety of plant there is a certain amount of moisture, degree of warmth, light intensity, etc., the *optimum*, which will induce the best development. With increase beyond the optimum, growth and development may be retarded until the *maximum* is reached, beyond which death results; or with reduction below the optimum, life processes may be slowed up until they either reach a very low ebb or cease entirely.

3. Freedom from mechanical injuries.
4. Freedom from the depredations of parasites, either animals or plants.

It may be noted from this consideration of the needs or the requirements for a thrifty development that there are endless possibilities of disease production. Any given plant must be inherently sound; must have the proper degree of heat, intensity of light, amount of air and food materials; the proper physical environment and freedom from mechanical injuries or the inroads of parasites, if it is to make a satisfactory growth. If any one of these factors, or a group of these factors, is seriously disturbed, disease in either mild or severe form will result. There may be a disturbed water relation—too little or too much—a too great intensity or duration of light, too low or too high temperatures, deficiencies or excesses in chemical elements or compounds, or parasites that are simply living their own life may cause disturbances in the physiological processes of the hosts from which they are obtaining their food.

Kinds of Plant Diseases.—Three great groups of plant troubles may be recognized, according to the nature of the causal agents: (1) *non-parasitic* disturbances, due to lack of proper inherent qualities, to improper environmental conditions of soil or air and to injurious mechanical influences; (2) *virus diseases*, due to an infectious principle, a so-called *virus*, which can be transmitted from one plant to another; (3) *parasitic troubles*, due to the inroads of other organisms, or parasites, that live at the expense of their hosts, or suscepts, and cause a slight or pronounced disturbance in their life processes.

Non-parasitic Diseases.—A proper understanding of the physiological processes in plants, especially the relation of these to production, would therefore seem of paramount importance in shedding light on the numerous non-parasitic diseases. In studying this group of diseases, the farmer, gardener or professional plant doctor can profit by being well grounded in fundamental physiological principles and in the recognized practices of scientific agriculture and horticulture. This phase of plant-disease study must then stand in intimate relation to the work of the physiologist, the agronomist, the horticulturist and the soil chemist and physicist. Within recent years, the rapid development of plant pathology has given greatest emphasis to the diseases due to parasites. Textbooks in the English language like those by Duggar, Butler and others have really dealt with bacterial and fungous diseases, either alone or with but slight emphasis on the non-parasitic disturbances. The drift has been away from the standard set by such workers as Sorauer and Frank in Germany, each of whom gave about equal emphasis to parasitic and to non-parasitic diseases. The proper relative importance of the two types of plant diseases is very well measured by the treatment of the troubles of the apple and the potato in recent bulletins. Examples of these non-

parasitic troubles may be cited, as the bitter pit, Jonathan spot, scald, cork, drought spot, water core and winter sun scald of the apple; or the blackheart, frost necrosis and internal brown spot of the potato. All these are as truly diseases as though they were caused by the presence of a form of animal or plant life.

Virus Diseases.—This is a group of somewhat related diseases in which the disturbed condition is the result of an infectious principle, a so-called "virus," which can be transmitted from diseased to healthy plants and communicate the disease. These troubles agree with the parasitic diseases due to bacteria and fungi in being infectious, but no visible organisms or causal agents are known. The infective principle, whatever it may be, is present in the juice or cell sap of a diseased plant, the different diseases showing varying degrees of infectiousness. In the extreme, they may be transmitted by mere contact, while in others, less infectious, organic union of a diseased and healthy plant by grafting is necessary for the communication of the disease. The virus diseases behave so much like germ diseases that there has been a common theory that they are due to invisible microorganisms, much smaller than the smallest bacteria.

The virus diseases are of the systemic type, the entire body of a sick plant being pervaded by the infectious principle. Under natural conditions, insects are the most important agents in the transmission of the virus from one plant to another. The first virus disease to be given definite recognition, the mosaic of tobacco, was reported in 1886. Since that time, numerous diseases of the virus type have been described as affecting both wild and cultivated plants. The demonstrations as to the behavior of the virus diseases and the important part which they play in crop losses constitute one of the most important achievements of modern phytopathology.

Parasitic Diseases.—In the broadest use of the term, parasitic diseases of plants should include all disturbances in the life and production of plants due to the attacks of some other living organism, either animal or plant. The attacking organisms which have become robbers or unbidden guests, living at the expense of plants, are the *parasites*, while the plants which harbor and entertain these robbers are the *hosts*. This is not the common society concept of the word "host," but it is a convenient term and has been established by usage. "Suscepts" has been offered as a substitute (Whetzel *et al.*, 1925). In order that we may understand the field covered by plant pathology, a brief synopsis of the groups of organisms which prey upon plants, either wild or cultivated, may be presented: (1) animal parasites or pests; (2) plant parasites.

The Animal Parasites.—These pests are furnished by five great groups: (1) the *higher animals*, including gophers, squirrels, mice, rabbits or other mammals; (2) the *arthropods*, including a limited number of

crustaceans like the sow bugs, a few myriapods or thousand-legged worms, a larger number of arachnids, represented by mites and red spiders, and an enormous number of hexapods, or true insects; (3) *mollusks*, represented mainly by snails and slugs; (4) *vermes* or worms, represented by certain genera of nematodes or eelworms; and (5) *protozoa*, represented by certain rhizopods and infusoria.

The Plant Parasites.—Those parasites which belong to the plant kingdom are found in five different groups: (1) the higher or *seed plants* (spermatophytes), as illustrated by the dodders or love vines (*Cuscuta spp.*) on clover, alfalfa and various other hosts; the leafy mistletoes (*Viscum* and *Phoradendron spp.*) on various fruit, forest or shade trees; the dwarf or scaly mistletoes (*Razoumofskya spp.*) on coniferous trees; the broom rapes (*Orobanche spp.*) on tobacco, clover and a few other hosts; and other parasites of lesser economic importance; (2) the *algæ*, including only a very few species of little economic importance; (3) the *fungi*, of first importance as the cause of infectious or contagious diseases, as illustrated by molds, blights, rots, mildews, rusts, smuts, etc.; (4) the *bacteria* or Schizomycetes, with a large and ever-increasing number of infectious diseases to their credit. These plant parasites listed in the order of their importance as causal agents of disease are: fungi, bacteria and seed plants. Of the thousands of recognized species of fungi, by far the larger number are scavengers, living on dead or decomposing organic remains, but a large number have become confirmed parasites or are able to live parasitically when the opportunity offers. So numerous are these parasites that nearly every plant, wild or cultivated, has one to several, or in some cases numerous, fungi which prey upon it.

Province of Plant Pathology.—It must be evident that the consideration of all parasitic troubles would afford a field so broad and would demand such a diversity of training that it could hardly be adequately mastered by the plant doctors. Specialization, or the division of labor, has somewhat restricted the province of the plant pathologist or professional plant doctor. Consideration of the depredations of higher animals and their effects upon agricultural production is generally assigned to the *economic zoologist*. Study and investigation of the insect pests of economic plants, using the term "insect" in its broadest sense rather than its strictly scientific meaning, to include all arthropods, constitute a very important part of the field of the *economic entomologist*. The field of the plant pathologist, as it is generally recognized at present, includes the consideration of all non-parasitic diseases, the virus diseases, all troubles due to the four groups of plant parasites and, in addition, the nema diseases, or those due to the eelworms or nematodes and also those of protozoan origin. A few years ago, no protozoan parasites of plants were known, but recent investigations point to protozoans as a group of increasing importance as furnishing the causal agents of disease. The

establishment of the pathogenicity of bacteria for man and domestic animals antedated the recognition of specific bacterial diseases of plants. Many protozoan parasites of man and animals are recognized today, some as very important agents of disease, but so far as protozoan diseases of plants are concerned we are now entering a field of investigation the importance of which only the future can reveal.

The problems with which the professional plant doctor must deal call for the broadest kind of a training. He must be well grounded in the fundamentals of pure botany, including plant physiology, histology and taxonomy of seed plants; on account of the importance of fungous diseases, a detailed knowledge of systematic mycology is an essential part of his equipment; the increasing importance of bacteria as producers of plant disease would make him something of a bacteriologist; the similarity of plant troubles to those caused by insects leads him into the field of economic entomology; without a thorough training in chemistry and physics, he could make but little real progress; since the final goal of the plant doctor is the prevention of crop losses, or the destruction of plants or plant products, he must be in sympathy with agriculture in general and should have a good working knowledge of agronomic, soil and horticultural science; and, finally, in order that the researches of the world may be an open book to him, the ability to read Latin, German and French should be acquired.

The Beginnings of Plant Pathology.—It is undoubtedly true that plant diseases were robbing the tiller of the soil of some of the fruits of his labor at the very dawn of man's civilization. Early historic and religious writings record the blighting, blasting, rusting or mildewing of the crops of the ancient peoples. Throughout the ancient era and the Dark Ages, but little was known of the true physiological behavior of plants; consequently, the ideas concerning plant diseases remained fragmentary and unorganized. Superstitions, dogmas and false interpretations of phenomena characterized the period. Even in the seventeenth and eighteenth centuries, there was but a slow dawning of our modern ideas of disease, and while the early part of the nineteenth century saw the publication of two epoch-making works ("Exantheme der Pflanzen," by Unger in 1833, and "Pflanzen-Pathologie," by Meyen in 1841), neither author had knowledge of our current idea of the parasitism of fungi. This is clearly shown by the fact that the later writer attributes the smut of corn to the stagnation of sap brought about by excessive and unnatural fertilizing and calls the smut spores the product of an abnormal nutrition. The period from the early beginnings of civilization to 1853, with its slow advancement, may be called the "formative era of plant pathology."

The Early Modern Era.—This was ushered in by the publication, in 1853, of the classical work of Anton De Bary, the great German botanist and mycologist, on "Die Brand Pilze," in which he established the

parasitism of the fungi associated with rusts and smuts. The causal relation of *Phytophthora infestans* to the late blight of potatoes was proved by his investigations published in 1861, and the establishment of the relation of the æcidium on the barberry to the rust fungus on wheat followed in 1865. The brilliant mycological work of the Tulasne brothers and others in France on the life history of ergot, powdery mildews, rusts and smuts (1847–1863) and the contributions of M. J. Berkeley on British fungology and vegetable pathology (1846–1860) are also landmarks in the early part of the modern era. It remained, however, for Julius Kühn, a contemporary and countryman of de Bary, to produce the first modern textbook of plant pathology—one based on the mycological discoveries of his contemporaries. This work, entitled "Die Krankheiten der Kulturgewächse, ihre Ursachen und ihre Verhütung," appeared in 1858, and presents such an accurate, concise and complete treatment of phytopathology that Kühn may with justice be given the title of "father of modern plant pathology." Soon after this, the "brilliant researches and convincing demonstrations of that noted French savant Louis Pasteur swept away the nebulous foundations of spontaneous generation" (1860–1864), and improvement in culture methods soon followed which made possible more accurate studies of the life histories of fungi and the etiology of disease (Whetzel, 1918).

The Place of Germany in the Early Modern Era.—The time was now ripe for more rapid advances in plant pathology, and important investigations soon appeared and manuals were published which were destined to mold and direct the progress of plant pathological knowledge. At this time, the most important contributions were made by German scientists, a few of the outstanding figures being Brefeld, to be credited with numerous publications of a mycological character, beginning in 1872; Sorauer, noted as the author of "Handbuch der Pflanzenkrankheiten," first published in 1874 as a single volume but in its present edition expanded to six volumes; Hartig, the "father of forest pathology," and the author of "Wichtige Krankheiten der Waldbäume" (1874) and other treatises on general and forest pathology; Frank, the author of "Die Krankheiten der Pflanzen," first published as a single volume (1880) but later expanded to three volumes (1895–1896), and also of numerous other botanical contributions; Kirchner, whose work entitled "Die Krankheiten und Beschädigungen unserer landwirtschaftlichen Kulturpflanzen" (first edition, 1890; third edition, 1923) was arranged by hosts for more practical use; von Tubeuf, author of "Pflanzenkrankheiten durch Kryptogame Parasiten verursacht" (1895), later translated by Smith as "Diseases of Plants Produced by Cryptogamic Parasites" but more properly "Cryptogamic Parasites as the Cause of Disease" because of its strong mycological trend; Küster, who produced the first comprehensive work on pathological plant anatomy, "Pathologische Pflanzenanatomie"

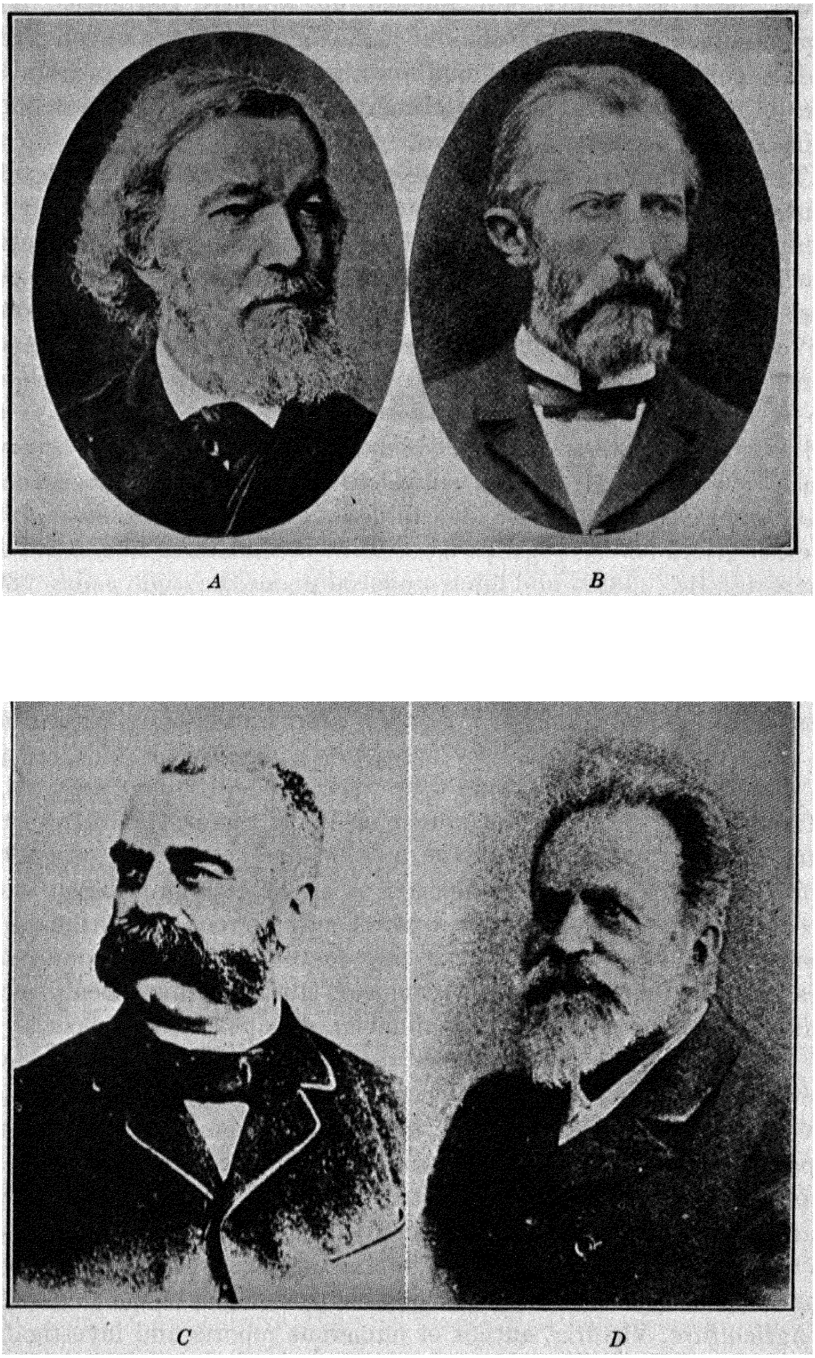

FIG. 1.—Four noted German plant pathologists. *A*, Julius Kühn; *B*, Anton De Bary; *C*, Robert Hartig; *D*, Paul Sorauer. (*From illustrations in "History of Plant Pathology," by H. H. Whetzel.*)

(1903), later translated into English by Frances Dorrance. While only prominent men responsible for manuals or texts are here recorded, credit should be given to numerous investigators who, by their careful and painstaking researches, placed Germany far in the lead in the realm of plant pathology.

The Contribution of Other Foreign Countries.—While this development of the science of plant pathology was proceeding in Germany, the scientific workers in other countries were making important contributions which were reflected in various manuals and textbooks. A few of the more noteworthy may be recorded: Prillieux and Delacroix in France, the former the author of "Maladies des Plantes Agricoles et des Arbres Fruitiers et Forestiers" (1895), a comprehensive work in two volumes; the latter the author of "Maladies des Plantes Cultivées" (1902), "Maladies Non-parasitaires" (1908) and "Maladies des Plantes Cultivées dans les Pays Chauds" (1911), completed after his death by his successor Maublanc; Comes, Savastano and Berlese in Italy—Comes, author of an extensive work on cryptogamic parasites, "Le Crittogame parassite delle piante agrarie" (1882) and later produced under the same name (1891); Savastano, who began publishing in 1881 and later produced his book on the diseases of trees, "Patologia arborea applicata" (1910); and Berlese, best known for his publications on fungi which began in 1883, the most noteworthy being "Icones Fungorum omnium hucusque cognitorum ad usum Sylloges Saccardianæ accommodatæ" (1894–1905), designed to supplement the descriptions of Saccardo's *Sylloge Fungorum;* J. Ritzema Bos in Holland, best known as one of the editors of the Dutch journal on plant diseases, *Tijdschrift over Plantenziekten* (begun in 1904); Woronin, mycologist and pathologist of St. Petersburg, Russia, known especially for his very creditable work on the club root of cabbage (1878); Rostrup, the most noted Danish phytopathologist, who produced his greatest contribution, "Plante patologi" (1902), in his seventy-second year; Eriksson, a prominent Scandinavian scientist, known especially as the originator of the "mycoplasm" theory and as the joint author with Henning of an important work on cereal rusts (1896), "Die Getreideroste, ihre Geschichte und Natur sowie Massregeln gegen dieselben"; Ideta in Japan, who published a book translated into German under the title "Lehrbuch der Pflanzenkrankheiten in Japan" (1903); H. Marshall Ward in England, author of many researches and also known because of his two books "Timber and Some of Its Diseases" (1889) "Diseases of Plants" (1901); and McAlpine, vegetable pathologist of the Department of Agriculture, Victoria, author of numerous reports and investigations but familiar to present pathologists from his "Fungous Diseases of Stone Fruits in Australia" (1902), "The Rusts of Australia" (1906), "The Smuts of Australia" (1910) and five rather elaborate reports on "Bitter-pit Investigations" (1911–1916). This gives a brief survey of a few of

the most eminent phytopathologists of the countries mentioned. Many worthy investigators are of necessity omitted, and the student is referred to the literature cited for more complete details.

Phytopathology in America.—An account of the rise and development of plant pathology in America may now be presented. This subject was first taught incidentally with botany by Burrill in 1873 at the University of Illinois and as a special subject by Farlow in 1875 at Harvard. Two early events which gave impetus to the development of phytopathological investigations in the United States were: first, the organization of a Section of Mycology in the Division of Botany of the U. S. Department of Agriculture (1885), with Lampson-Scribner as first Federal phyto-

FIG. 2.—Class in plant pathology at the State College of Washington.

pathologist; and, second, the organization of the state agricultural experiment stations by the Hatch Act of 1887, which provided $15,000 annually to each state experiment station for "scientific investigation and experiment respecting the principles and application of agricultural science," with a further addition of $15,000 by the Adams Act of 1906 for more fundamental researches bearing upon agriculture. Increased emphasis has been given to the phytopathological investigations as a result of the Purnell Bill, passed by Congress in 1925, which has given $60,000 additional Federal funds to each experiment station.

The Development of Plant Pathology in the U. S. Department of Agriculture.—With the gradual increase in financial support to the U. S Department of Agriculture, the work in plant pathology has grown from its simple beginnings with its single worker to many divisions in the present Bureau of Plant Industry, with a whole army of plant doctors whose activities reach the remotest corners of the country. Plant-disease investigations in this bureau are provided for in the following

administrative groups: (1) Horticultural Crops and Diseases including Fruit and Nut Diseases, Truck and Ornamental Diseases, Pathological Laboratory (mainly bacterial diseases) and Fruit and Vegetable Handling, Transportation and Storage Investigations; (2) Cereal-pathology Investigations in Office of Cereal Crops and Diseases; (3) Forage Crops and Diseases; (4) Forest Pathology; (5) Sugar-beet Pathology, Curly-top Investigations and Sugar-cane Pathology in Office of Sugar Plants; (6) Tobacco-disease Investigations in Office of Tobacco and Plant Nutrition; (7) Nema Diseases in Office of Nematology; (8) Mycology and Disease Survey including the Mycological Collections and the Plant-

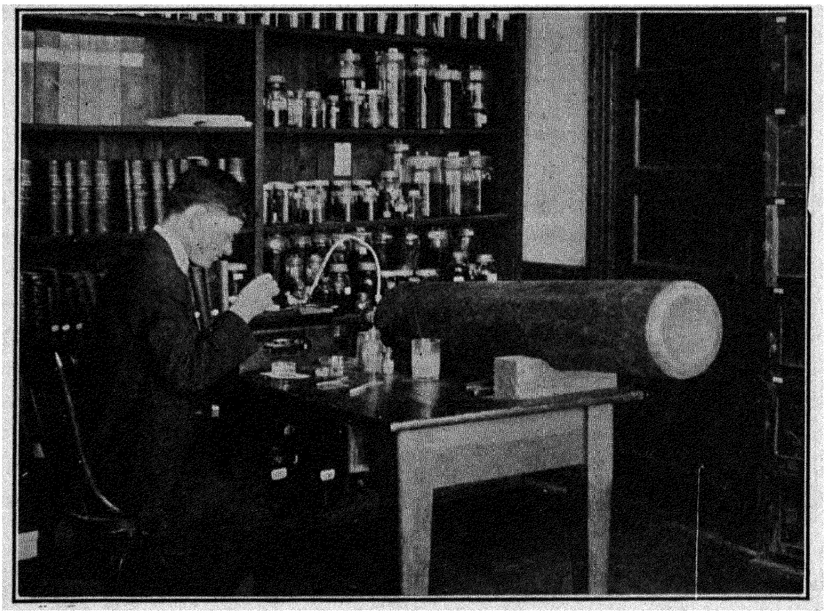

FIG. 3.—Preparing sections for diagnosis of a disease by the use of the freezing microtome. B. F. Dana, formerly assistant professor of plant pathology, State College of Washington.

disease Survey; (9) Blister-rust Control; (10) Barberry Eradication; (11) Citrus-canker Eradication; and (12) Phony-peach Eradication.

In addition to these offices in the Bureau of Plant Industry, some plant pathological interests are provided for in other bureaus: (1) Fungicides by the Plant Pathological Unit of the Food and Drug Administration and the Insecticide Division of the Bureau of Chemistry and Soils; (2) Quarantines under the Plant Quarantine and Control Administration; (3) Inspection of Fruits and Vegetables in Division of Fruits and Vegetables of Bureau of Agricultural Economics; and (4) Office of Cooperative Extension Work in Extension Service.

The Development of Plant Pathology in the Various States.—The Hatch Act creating the agricultural experiment stations placed them

with the agricultural colleges of the different states; consequently these institutions have been the state centers of plant pathological activity, in many cases cooperating with the Federal departments. This direct support and encouragement to teachers and investigators from the land-grant colleges and by the Federal department have made possible rapid strides in our knowledge of plant diseases. At first, teaching and investigation in plant pathology went hand in hand and were provided for as a part of the work of the departments of botany. In many states, teaching and investigation have now been separated, or separate departments

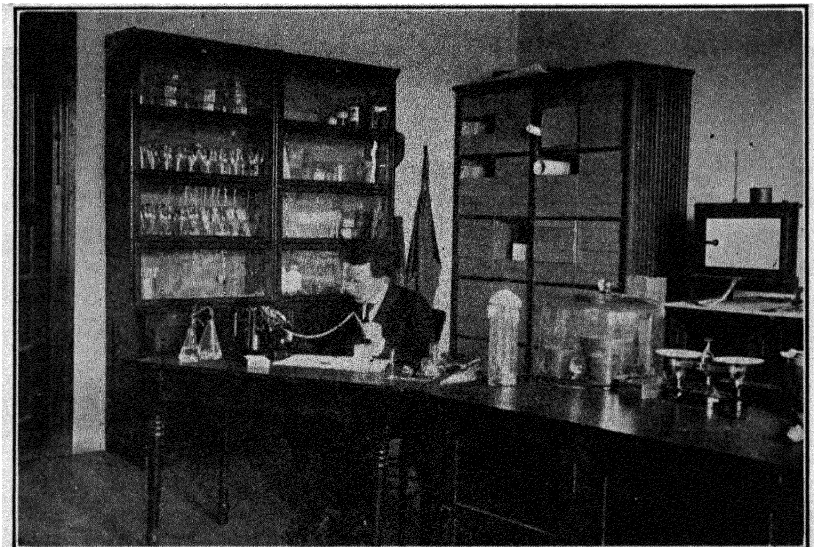

FIG. 4.—Preparing paraffin sections for the study of a plant disease. Wheat-smut studies by H. M. Woolman.[1]

of plant pathology organized. Whatever the organization, there are one to several workers in each agricultural experiment station devoting a part or all of their time to the investigation of plant-disease problems, and plant pathology has become one of the recognized subjects in the curriculum of the agricultural college.

The Smith-Lever Bill, passed by Congress in 1914, and the Capper-Ketcham Act passed in 1929, which provided funds for extension work in agriculture, have made possible the carrying of plant-disease information directly to the people by plant-disease specialists. Many states have already adopted this plan and are using one to several extension plant pathologists who work in cooperation with the investigators of the experiment stations.

Various states are maintaining well-organized state departments of agriculture, and plant-disease work is receiving more and more attention. While police or inspection duties are of first concern in the state depart-

[1] Deceased.

ments, some attention is given to field phases of research and to educational features. Horticultural inspectors should be well trained both in plant pathology and in economic entomology, a precaution which up to the present has not always been taken; but undoubtedly this state service will soon show improvement.

It is significant that but little of the real progress in plant pathology has come from within the walls of our great endowed universities or state universities separate from the land-grant college. Within the last few years, these institutions have begun to introduce instruction in plant pathology and to give more encouragement to plant-disease investiga-

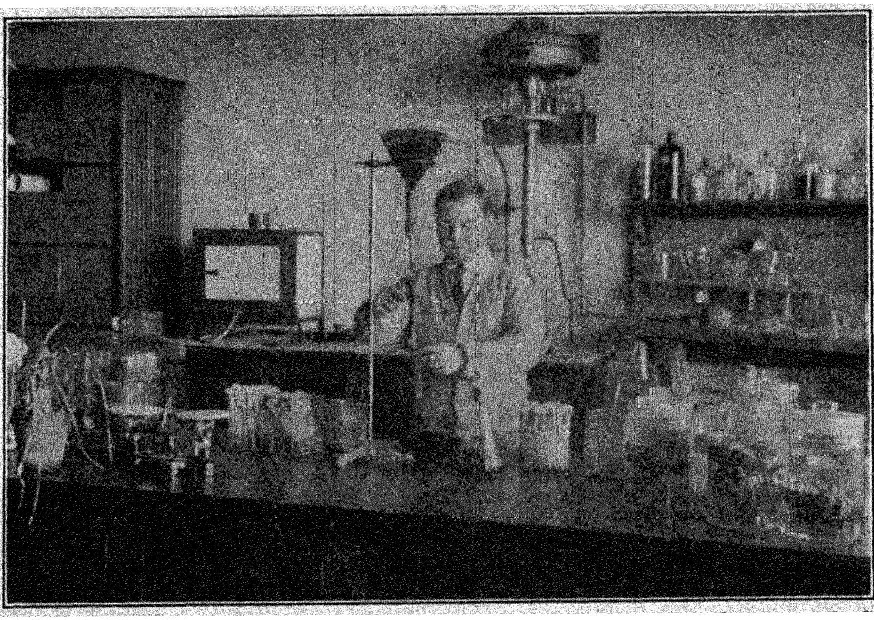

FIG. 5.—Preparing culture media for the study of bacterial or fungous pathogenes. D. C. George,[1] plant pathologist, Arizona State Department of Agriculture, Phoenix.

tions. This is an omen of still further progress for the science of plant pathology, which has today reached a higher development in America than in any other part of the world. "Science like empire marches ever westward," and the center of phytopathologic science has been transferred from the Old to the New World.

American Contributions.—In the early teaching of plant pathology in America, principal reliance was placed upon the German manuals and texts or English translations of these works. American workers have been so busy with experiment-station activities or investigations that they have had little time for the writing of books, with the result that many of the most noted pathologists of our country are known only from their published researches. The mills have been grinding steadily, and

[1] Deceased.

Federal and state bulletins and scientific periodicals are teeming with plant-disease information. The rapidity of our progress has militated against the production of textbooks, for the orthodox lecture of today is out of date tomorrow.

Those who were attempting to use von Tubeuf and Smith's "Diseases of Plants Induced by Cryptogamic Parasites" welcomed "Minnesota Plant Diseases," by Freeman, in 1905, and "Fungous Diseases of Plants," by Duggar, in 1909. The "Spraying of Plants," by Lodeman, also published in 1909, was a welcome contribution to our knowledge of that important method of disease control. "Diseases of Economic Plants," by Stevens and Hall (1910 and 1921), was the first American text to include any non-parasitic diseases, but in this they were given but little emphasis. Symptoms, effects and control were the features emphasized

FIG. 6.—A field demonstration of potato diseases by G. L. Zundel, formerly extension specialist, State College of Washington. (*Photograph by F. C. Meier.*)

for the parasitic diseases, so the student who used this book would learn but little about fungi. "Bacteria in Relation to Plant Diseases" (1905), by Smith, began a new chapter in our knowledge of bacterial diseases and has been followed by two other large volumes (1911 and 1914). A single profusely illustrated volume on "Bacterial Diseases of Plants" has since been published by the same author (1920). Stevens' work on "The Fungi Which Cause Plant Diseases" (1913) and "Plant-disease Fungi" (1925), Clements' "Key to the Genera of Fungi" (1909) and Clements and Shear's "The Genera of Fungi" (1931) have been of special value to plant pathologists as well as to mycologists. Mention should be made of two general books: "Diseases of Tropical Plants," by M. T. Cook (1913), and a "Textbook of Mycology and Plant Pathology," by Harshberger (1917). Several manuals devoted to the diseases of special

groups of plants have appeared: "Manual of Fruit Diseases," by Hesler and Whetzel (1917); "Manual of Tree Diseases," by Rankin (1918); "Diseases of Truck Crops and Their Control," by Taubenhaus (1918), and a modification of this with some additions by the same author under

FIG. 7.—American authors of books on plant diseases. *A*, Charles Chupp, Cornell University; *B*, Melville Thurston Cook, Insular Experiment Station, Rio Piedras, Porto Rico; *C*, Benjamin Minge Duggar, University of Wisconsin, *D*, Edward Monroe Freeman, University of Minnesota; *E*, John William Harshberger,[1] University of Pennsylvania; *F*, Lexemuel Ray Hesler, University of Tennessee; *G*, Charles Elmer Owens, Oregon Agricultural College; *H*, William Howard Rankin, New York (Geneva) Experiment Station; *I*, Erwin Frink Smith,[1] U. S. Department of Agriculture; *J*, Frank Lincoln Stevens,[1] University of Illinois; *K*, Jacob Joseph Taubenhaus,[1] Texas Agricultural Experiment Station; *L*, Herbert Hice Whetzel, Cornell University. (*From photographs received from the several authors.*)

the name of "Diseases of Greenhouse Crops and Their Control" (1919); "Manual of Vegetable-garden Diseases," by Chupp (1925); and "Outlines of Forest Pathology," by E. E. Hubert (1931).

[1] Deceased.

Several general bulletins issued by state experiment stations are worthy of special mention: "A Brief Handbook of the Diseases of Cultivated Plants in Ohio," by Selby, first published as *Bul.* **121** (1901) and revised as *Bul.* **214** (1910); "California Plant Diseases," by R. E. and E. H. Smith (*Cal. Bul.* **218,** 1911); and "The Parasitic Fungi of New Jersey," by Carl A. Schwarze (*N. J. Bul.* **313,** 1917).

Landmarks of Plant Pathology.—A few of the outstanding discoveries or events which have marked the progress of modern plant pathology may now be outlined:

1. Proof of the parasitism of fungi as presented in the researches of the German botanist Anton De Bary on the rust and smut diseases (1853).

2. Proof of the heterœcism of the rusts as illustrated by the relation of the æcidium on the barberry to the red- and black-rust stages of the stem rust on wheat. This was another of De Bary's important contributions to mycological and pathological science published in 1864.

3. The perfection by Robert Koch (1881) of the plate method of isolating bacteria and fungi as a substitute for the cumbersome and rather unsatisfactory "fractional" method of Klebs and the "dilution method" of Lister. Koch substituted a gelatin medium which was solidified on glass plates. Agar was soon added and Petri dishes were substituted for glass plates, but the term "plate cultures" was retained. This improvement in isolation technique made possible more rapid progress in the study of life histories of many fungi and greatly facilitated the demonstrations of the pathogenicity of both bacteria and fungi.

4. The proof of the relation of bacteria to the fire blight of the apple and pear. This is to be credited to Thomas Burrill, professor of botany in the University of Illinois (1879–1881). It is significant that at this time, botanists in other parts of the world were also directing their efforts to the study of bacteria in connection with plant diseases, as shown by the work of Prillieux in France on the rose-red disease of wheat (1879), Wakker in Holland on the yellow disease of hyacinths (1883–1889) and Comes in Italy on bacterioses of several hosts (1880). America is proud of this notable contribution of Dr. Burrill that established beyond contradiction the fact that a definite species of bacterium, *Bacillus amylovorus*, was the causal agent of fire blight.

5. The discovery, by Millardet in France, of the effectiveness of copper and lime as an agent in the control of downy mildew of the grape and the introduction of our well-known Bordeaux throughout the vineyards of France (1882–1885). The use of this fungicide in America for the control of late blight of potato and the scab of apple soon followed, and it rapidly came into very general use as the panacea for various plant ills. The Bordeaux period held sway until the discovery of lime sulphur, which proved more satisfactory than Bordeaux for many crops.

6. The discovery, by Jensen of Denmark, of the effectiveness of the hot-water treatment of cereals in the control of their smut diseases (1887).

7. The introduction of formaldehyde as a disinfectant. Although discovered by Hoffman, a German chemist, in 1867, it was not used as a disinfectant until 1888 (Trillat), and Bolley of North Dakota first demonstrated its practical application as a fungicide in the control of seed-borne diseases (1897).

8. The revival of the use of lime sulphur as a combined insecticide and fungicide by the demonstration of its effectiveness as a summer spray in the control of apple scab. This is to be credited to Cordley of Oregon and Piper of Washington (1906–1908), and their discovery was soon taken up by the pathologists of the eastern United States. Lime sulphur and lead arsenate rapidly replaced Bordeaux and Paris green as the standard apple spray.

9. The demonstration of the cause and nature of crown gall by Dr. Erwin F. Smith and C. O. Townsend, of the U. S. Department of Agriculture (1907). The proof that the crown gall of cultivated fruit trees and other plants was due to a specific bacterial organism, *Pseudomonas tumefaciens*, was the second great event in the history of bacterial diseases to be credited to America. This discovery left the myxomycete *Dendrophagus globosus* in its supposed relation to crown gall as one of the wrecks that mark the pathway of scientific progress.

10. The organization of the American Phytopathological Society at the Baltimore meeting of the American Association for the Advancement of Science in 1909. This society and the journal *Phytopathology*, which was started in 1911 as its official organ, have been potent factors in stimulating and directing the trend of plant pathological work in this country.

11. The passage of the Simmons Bill by the Sixty-second Congress, by which the National Plant Quarantine Act became a law (1912). The enactment of this measure and the creation of a Federal Horticultural Board to administer the act constituted the first legalized, national effort aimed to exclude foreign insect pests and plant diseases from this country. Under this law, numerous quarantines, both domestic and foreign, have been established.

12. Proof of the effectiveness of dusting with finely powdered fungicides and insecticides as a substitute for spraying. Finely ground sulphur and powdered lead arsenate were used with success by various pathologists at Cornell University (1913–1917), and more recently copper dusts have been successfully employed. By the recognition of the fact that fineness of a dust fungicide is a measure of its efficiency and by the perfection of improved dusting machinery, dusting as a method of disease prevention has become an accepted practice for certain crops and diseases.

13. The recent rapid increase in our knowledge of the so-called virus diseases of plants, including mosaics, leaf-roll and peach-yellows types. From first proof of the mosaic of tobacco as an infectious disease (1888), our knowledge of this group of diseases made slow progress until the last few years, during which numerous new troubles of various crop plants have been recognized and increased importance has been attached to the rôle of insects in the transmission of the virus or active principle. If the present activities in this field of plant pathology continue, future historians may well designate the present time as the *mosaic period*.

14. Three epiphytotics of recent times have had a pronounced influence upon the interest of the general public in plant-disease problems and have also been a stimulus to phytopathologists. Large sums of money have been appropriated for investigating these diseases and for carrying out eradication or control measures. These diseases are as follows: (1) the *chestnut-tree blight*, the most virulent and devastating disease of a forest tree that has ever been recorded (1906 to present), which threatens to exterminate our American chestnut from its native haunts; (2) the *blister rust of white pine*, a heterœcious rust, which passes part of its life cycle on currants or gooseberries and is capable of spreading from these hosts to the white or other five-needle pines—it has already spread to an alarming degree in the northeastern United States (mainly since 1915) and has more recently appeared (since 1921) in British Columbia, Washington and Idaho; (3) *citrus canker*, a bacterial disease, which after its introduction (about 1909–1910) spread from Texas to the other Gulf states and by 1914 was epiphytotic in portions of the citrus districts of the South. All of these diseases were undesirable immigrants from foreign countries: chestnut blight from China, blister rust from Europe and citrus canker from Japan. The devastation from these diseases is the cause of alarm, and they have served to emphasize the constant danger from the introduction of foreign parasites.

15. The production of immune or resistant varieties of plants by breeding and selection is the most modern phase of plant-disease control. The primary stimulus came from the work of Orton in breeding strains of cotton, cowpeas and melons resistant to Fusarium wilt (1899–1909). With the increased emphasis on breeding in relation to agriculture, plant breeders are becoming pathologists and pathologists are becoming plant breeders.

16. The campaign for the eradication of the barberry as a means of preventing the epiphytotics of stem rust of wheat has been actively prosecuted since 1917 by the Federal department in cooperation with the various states of the northern Mississippi Valley. This was first made possible by the generous appropriations to the U. S. Department of Agriculture for the increase of food production as a prominent war meas-

ure, and this financial support has been continued, largely because of the reported success of similar measures in Denmark.

17. The introduction of the copper carbonate dust treatment of wheat for bunt or stinking smut is a recent practice that has been widely adopted in every wheat-growing state in this country as well as in foreign lands. The treatment was originated by Darnell-Smith in Australia in 1915, but more extensive experiments have been carried out in California, Washington (1921 to present) and other states. Several seed-dusting machines have been developed on a commercial scale in Washington and California, and as a result the dust treatment is more extensively used on the Pacific Coast than in the land of its origin.

18. The discovery of the function of the pycnia in rusts (Craige, 1927–1931) followed by demonstrations of the origin of physiologic strains by hybridization and mutation in the rusts and also in various other parasitic fungi by several investigators in the United States, Canada and Australia.

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CHAPTER II

SYMPTOMS OF DISEASE IN PLANTS

It is highly important that all persons engaged in general farming, truck gardening, fruit raising, floriculture or forestry should be able to recognize the presence of disease in plants. Every plant in its particular way shows when it is suffering from disease. Crop producers should not only know how plants live and grow but should be familiar with those outward signs or symptoms which exist when plants are sick. The study of symptoms is the first step in making a diagnosis or the determination of the nature or identity of a trouble. A correct determination can be made in many cases by the observation of symptoms alone, but in others it is necessary to study environmental factors or to dissect diseased plants or portions of plants in the search for a pathogene.

In the nature of the resulting symptoms, there are no hard and fast lines that can be drawn between parasitic and non-parasitic diseases or between various "insect" troubles and those due to bacteria or fungi. The first thing of importance is to detect the presence of disease and then to endeavor to find out its cause, nature and probable outcome and the treatment or preventive practices which should be employed. The great diversity of symptoms, the numerous non-parasitic disturbances and the multitude of pathogenes so complicate diagnosis that the grower must frequently seek the services of the trained plant doctor.

Summary of Symptoms.—The following outline of symptoms will be of service to the practical grower and also to the plant-disease specialist:

1. Discoloration or change of color from the normal:
   A. Pallor:
      a. Pale green.
      b. Yellow:
         Etiolation.
         Chlorosis.
      c. Silvering.
      d. White—albinism.
   B. Colored spots or areas:
      a. General difference between parasitic and non-parasitic troubles; exceptions.
      b. Colors: Whitish or gray; red or purple; brown; black; variegated and concentrically zonate.
   C. Autumnal colorations or spring colorations.
2. Shot hole: Perforations of leaves:
   A. Physiological: Toxic substances; frost; drought; etc.
   B. Fungous or bacterial origin.

3. Wilting:
   A. Juvenile condition: "Damping-off" of seedlings.
   B. Adult plants:
      Physiological wilting with recovery.
      Wilting without recovery: "Wilt" diseases; thrombosis of woody plants.
4. Necrosis: Death of parts:
   A. Organs: Leaves, stems, flowers, fruits—blight.
   B. Localized areas of tissue: Internal necrosis, *e.g.*, bitter pit of apple; internal brown spot, net necrosis and blackheart of potato.
5. Dwarfing or atrophy—reduction in size:
   A. Entire individual: Non-parasitic; parasitic.
   B. Parts or organs: Leaves, fruits, etc.
6. Hypertrophy or increase in size of parts: Roots, stems, leaves, flowers, fruits:
   A. Increase in size of cells.
   B. Abnormal multiplication of cells or increase in number—Hyperplasia.
7. Transformation of organs or replacement of organs by new structures:
   A. Replacement: By sclerotium, as in ergot of grasses.
   B. Transformations of floral organs: Petals to foliage leaves, etc.; "phyllody."
8. Mummification: Transformation of fruits into shriveled structures, called mummies, which process is initiated by rotting, followed by resistance to decay. Mummies contain either dormant mycelium or overwintering fruits of fungi.
9. Alteration in habit and symmetry:
   A. Change of position from prostrate to erect or ascending.
   B. Change from rosette to cauline type.
   C. Change of simple leaves to lobed leaves.
   D. Reduction or increase in branching.
   E. Change in form of inflorescence.
   F. Change in symmetry of flowers.
10. Destruction of organs: *e.g.*, flowers or flower parts; seeds; seeds and pits in "bladder plums"; fruit (caryopsis) in cereals; inflorescence, as in loose smuts of cereals.
11. Dropping of leaves, blossoms, fruits or twigs.
12. Production of excrescences and malformations:
   A. Abnormal trichome growth—Erineum.
   B. Intumescences on leaves and stem blisters or swellings involving groups of cells but without the presence of a parasite.
   C. Pustules, warts, tubercles, galls or tumors. Parasite included. Minute to large and fleshy or woody; crown gall, etc.
   D. Cankers: Localized lesions generally resulting in corrosion of tissue with final production of an open wound, generally on woody structures:
      a. Parasitic.
      b. Non-parasitic.
   E. Witches' brooms.
   F. Hairy root: Simple; broom-root type; woolly-knot type; aerial form.
   G. Production of rosettes: Woody plants; herbaceous plants.
   H. Development of dormant or rudimentary structures or of new organs.
   I. Prolification.
   J. Rolling, curling or crinkling of leaves.
   K. Fasciation and spiralism.
   L. Roughening of surfaces: Local or extended.
   M. Deforming of fruits.
13. Exudations. Guttation and bleeding contrasted with formation of exudates:
   A. Bacterial exudates: *e.g.*, fire blight.

B. Slime flux: Deciduous trees; exudate semifluid, does not set into solid masses.
C. Gummosis: Product of decomposition of tissues; sets into solid masses, clear or amber colored. Common in citrus and stone fruits. Parasitic and non-parasitic.
D. Resinosis: Abnormal production of resin or pitch from coniferous trees.
E. Latexosis: Exudation of milky juice or latex.

14. Rotting:
   A. Dry rot and soft rot, the "gangrene" of plant tissue. Fleshy structures more generally show soft rot; woody structures, dry rot:
      a. Root rots: Fleshy or woody roots, *e.g.*, alfalfa, cotton; root crops—beets, carrots, etc.; woody roots—apple, cherry, etc., and shade or forest trees.
      b. Stem rots: Herbaceous stems—carnation, aster, potato; modified stems—tubers, rhizomes, bulbs or corms (storage organs); woody stems—dry rots, as *sap rot* or *heart rot*.
      c. Bud rots: *e.g.*, carnation and coconut bud rots, cabbage black rot.
      d. Fruit rot: Fleshy fruits of many kinds.
         (1) Non-parasitic: Blossom-end rot of tomato, blossom-end rot of watermelon.
         (2) Parasitic: Many on various hosts. Mostly fungi. Why few bacteria?

1. Discoloration or Change of Color from the Normal.—This deviation from the normal may be noted on various plant structures as illustrated by the pink coloration of the roots in pink root of the onion or by the red-spotted fruits characteristic of peach yellows, but the discolorations are very frequent and striking on parts which are normally green, such as herbaceous stems and foliage leaves. Plants, like people, frequently look pale when they are sick. Nutritional disturbances preventing the constant production of the green pigment chlorophyll cause normally green structures to become pale green or yellowish green or to exhibit pallor. The normal shade of green for any particular plant may be recognized, and the practiced eye soon learns to tell when something is wrong. The green pigment may disappear entirely and its place be taken by a yellow pigment. When this yellowing is brought about by lack of light or prolonged exposure to darkness, this condition is spoken of as *etiolation*, and the yellowed organs may be said to be *etiolated*. It may be noted in this connection that the etiolation or blanching of certain garden plants, such as asparagus, endives, artichokes, celery, etc., is practiced to secure desired flavor or tenderness and that in certain cases self-blanching varieties have been secured by selection. Somewhat similar effects may result from the operation of other factors, such as low temperatures, lack of iron, excess of lime, excess of alkali, presence of a virus disease like peach yellows or mosaic disease or from the disturbances caused by bacterial or fungous parasites. In these latter cases, it is the custom to speak of the condition as *chlorosis*, and the affected structures are said to be *chlorotic*. We speak, then, of iron or lime chlorosis, alkali chlorosis, infectious chlorosis, etc. Chlorosis as a disease symptom should not be confused with "panaschiering," in which the leaves are part green and

part yellow or spotted with white and green or white bordered. Plants showing these peculiarities have been selected and propagated for their ornamental value. Silvering of the foliage, or the assuming of a dull, metallic luster, is not uncommon in plums, apples and other woody plants and is a striking symptom of the infectious silver-leaf disease or may result from non-parasitic disturbances. Among plants, as among animals, there are the *albinos*, or those entirely devoid of pigment. Such constitutional albinos must soon succumb, since they have no power to produce carbohydrate food because of their lack of a chlorophyll apparatus.

Discolorations, instead of being general or diffuse, may show as more or less definite or circumscribed discolored spots or areas. In such cases, the spotting may result from localized disturbances from parasites or chemical agents or from deep-seated nutritional disorders in which

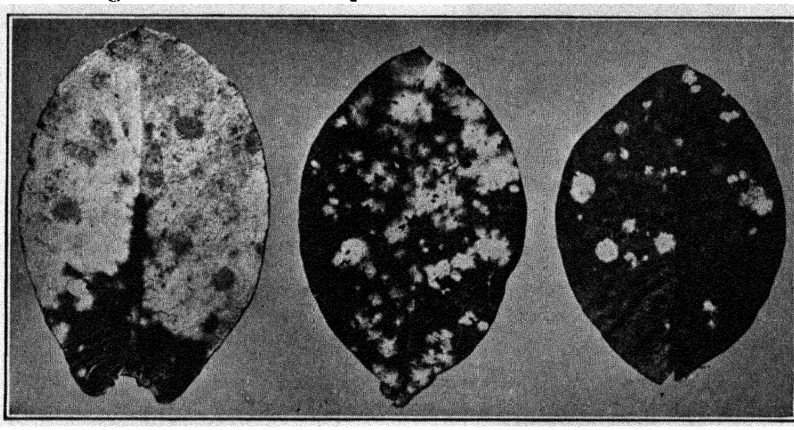


FIG. 8.—Powdery mildew (*Microsphæra alni*) of honeysuckle.

leaf spotting is but a phase. There is no certain character which will differentiate leaf spots of parasitic or non-parasitic origin, but many fungous infections, especially on netted-veined leaves, produce circular or subcircular areas or lesions which are soon limited in size. A spot comes wherever a spore of a pathogene lodges and is able to establish its mycelium; hence parasitic lesions are generally scattered irregularly over the surface of a leaf and show no definite arrangement with reference to the veins, either large or small. Apple foliage injured by arsenical spray containing free arsenic will sometimes show a brown spotting very similar to that caused by the black-rot fungus (*Physalospora malorum*). With numerous spotting, the lesions may coalesce to form more extended discolored areas. Some parasites that invade the leaf spread unchecked and so produce extended dead areas or may involve the entire leaf as in the late blight of the potato (*Phytophthora infestans*) or in leaf invasions by the bacteria of fire blight (*Bacillus amylovorus*). Leaf spots due to

certain kinds of non-parasitic disturbances are frequently located between the main veins of pinnately veined or palmately veined leaves. We can even note that these spots start at the blind ending of the veins in the smallest areolæ of the leaf or at points the most distant from the water-conducting channels. Drought injury or poisoning from smelter fumes may show this type of spotting, or, under other conditions, terminal or

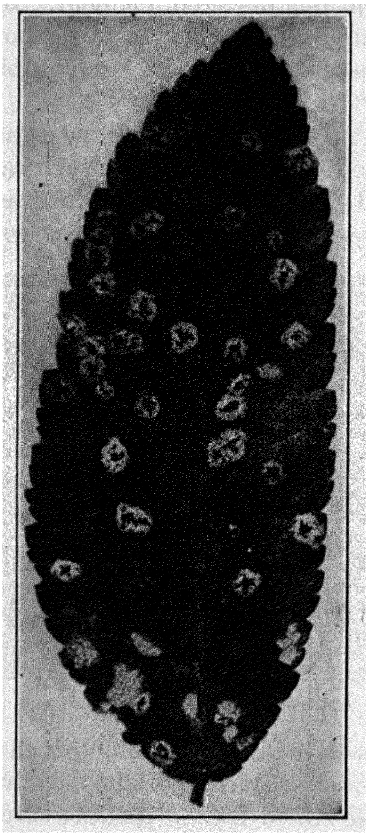


FIG. 9.—Leaf spot (*Gnomonia ulmea*) of elm.

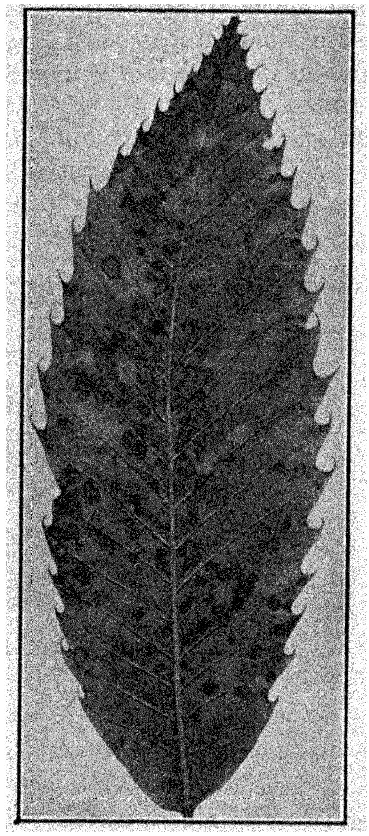
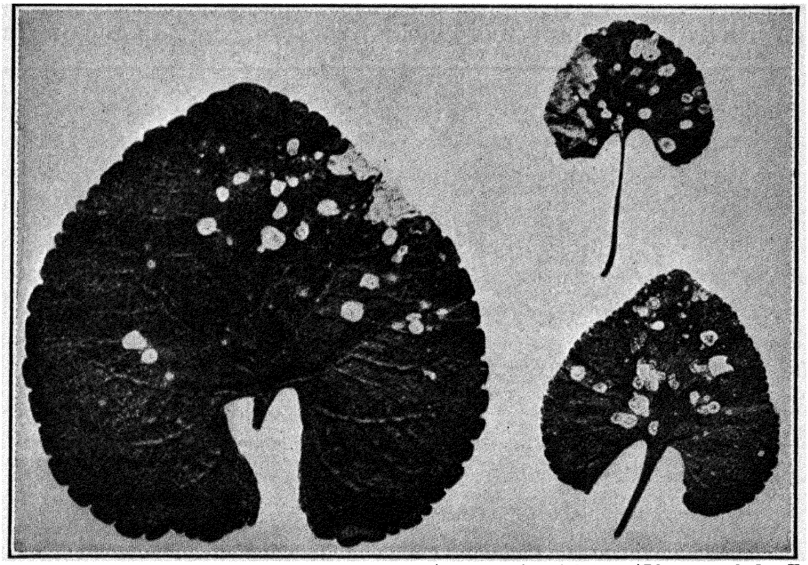


FIG. 10.—Leaf spot (*Marssonina ochroleuca*) of chestnut.

marginal discolorations may result, as in tip burn of the potato, a phenomenon of drought, or in poisoning of leaves by fumes of hydrochloric acid.

The discolored spots may be gray or whitish, yellow or orange, reddish or a shade of purple, brown, black or variegated and either of a solid color, concentrically zonate with shades of a single color, or of different colors on the two surfaces. The whitish coloration may be due to the superficial mycelium and spores, as in the powdery mildews of the apple, peach, cherry, grape, gooseberry, wheat and other grasses, lilac, honeysuckle, rose and many other hosts; to conidial sori, as in the white

rusts (*Albugo spp.*); to the groups of aerial conidiophores, as in the downy mildews of the grape, cucurbits, lettuce and also in various diseases caused by hyaline-spored imperfect fungi, as illustrated by species of Ramularia and Cercosporella; and finally to changes in the host tissue which lift the epidermis and admit air into the cells or intercellular spaces and so cause a reflection of the light. The centers of old leaf spots which were at first brown finally become a dull gray in various cases.



11.—Violet leaves affected with leaf spot (*Alternaria violæ*). (*Photograph by F. D. Heald and F. A. Wolf.*)

Yellow or orange spots may be due either to tissue changes or to the color of spore groups or masses. There may be a localized chlorosis, as in the leaf spot of the violet (*Alternaria violæ*), also in various downy mildews (*e.g.*, *Peronospora trifoliorum* on alfalfa) and in many rusts (*e.g.*, *Tranzschelia punctata* on peach), in which the sporulation is confined to the under surface. The spore fruits of rusts, especially the æcial or cluster-cup stages, and the paler forms of the so-called "red-rust" stage exhibit varied shades of yellow or orange.

Red or purple coloration in spots may also be due to tissue changes or to the masses of spores developed by the pathogene. In the first type, there is a decomposition of chlorophyll and the formation of red pigment (*anthocyanin*), which is dissolved in the cell sap. Chlorophyll disappears entirely or is masked by the presence of the red pigment. This red coloration is a host peculiarity and is frequently the first response of injured cells. Incipient infections may show the red coloration first, with change to brown later, while the occurrence of red or purple border may

characterize many more advanced lesions. The color variations are due in part to different degrees of acidity or alkalinity of the cell sap. Some of the rusts producing the more highly colored types of spore fruits would probably be classed as red (both aecial and red-rust stages), but in such cases more or less raised or powdery pustules of spores are evident.

Brown is the characteristic color of dead tissue and is the final color of the great majority of leaf spots, although some finally become grayish with age. Many rust lesions will show brown powdery pustules, representing either darker red-rust stages or paler black-rust sori. Superficial fungi with dark mycelium (sooty molds) or various dark-spored, imperfect

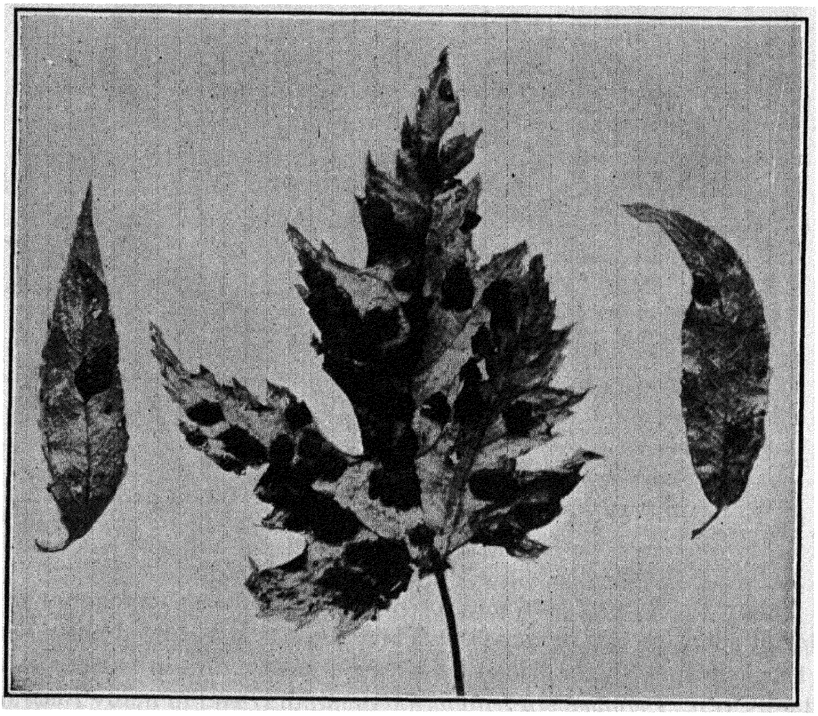


FIG. 12.—Tar spots (*Rhytisma spp.*) of maple and willow.

fungi may also cause the brown coloration due to the accumulation of mycelium or conidiophores or both (*Cladosporium fulvum* on tomato; *Venturia inæqualis* on apple).

The black coloration is well illustrated in the so-called tar spots (*Rhytisma spp.*) of the maple, willow and oak, the black spot (*Phyllachora spp.*) of grasses and other hosts, the black spot (*Diplocarpon rosæ*) of roses and especially in some covered and naked forms of black-rust sori (*Puccinia spp.* on cereals). In all of these examples the coloration is due to fungous tissue or to spores, but in a few cases the black spotting may be due to changes in the host tissue. This is well illustrated in the pear, in which injured leaf tissue frequently assumes a very dark or almost black

color (*Fabræa maculata*) or in the blackspot (*Bacterium pruni*) of plum fruits.

Leaf spots may show concentric bands of different shades of brown, as in the so-called target-board spots characteristic of early blight (*Alternaria solani*) of the potato or in the frog-eye spots (*Sphæropsis malorum*) of apple leaves. Concentric zones of different colors are a noticeable feature in the leaf spot (*Mycosphærella fragariæ*) of the strawberry, in which well-matured lesions are white or grayish in the center, surrounded by a brown zone bordered by purple and red, shading out into the surrounding healthy tissue. Black and gray or brown concentric zones alternate in the anthracnose (*Colletotrichum lagenarium*) of some squashes. Many leaf spots of hosts in which the red pigment is not commonly developed show a halo of yellow surrounding the brown, dead tissue. Some leaf spots show entirely different colors on the two surfaces, as is well illustrated in fruiting lesions of grape downy mildew which are yellow above and white below or in alfalfa downy mildew with yellow upper surfaces and grayish or faintly purple under surfaces.

The autumnal colorations of foliage may be briefly mentioned at this point. Chlorophyll is decomposed by the sunlight, and sometimes yellow and red colors of many and varied shades are formed, before the final assumption of the somber brown characteristic of dead-leaf tissue. The low temperatures which prevail check the normal life processes and prevent the reformation of chlorophyll. In a very similar way, the low temperatures of early spring are responsible for yellow or red coloration of young leaves.

2. Shot Hole or Perforation of Leaves.—The formation of localized lesions on the leaves is frequently followed by the falling out of the dead or diseased tissue, leaving circular or slightly irregular perforations, which have suggested the descriptive term "shot hole." Certain troubles have been called shot-hole diseases because of the peculiar effect upon the foliage. The occurrence of shot hole is not a diagnostic feature of certain species of fungi but is more properly to be viewed as a host peculiarity. Some varieties are prone to shot hole whenever localized areas of leaf tissue are killed, whether this death of tissue results from the presence of parasitic bacteria or fungi or from the operation of non-parasitic factors, such as toxic chemical agents, drought or frost. Among our stone fruits, the tendency to the shot-hole symptom is very marked, as in bacterial black spot (*Bacterium pruni*), the leaf spots or blights (*Coccomyces spp.*) of cherries and plums and the California blight (*Coryneum beijerinckii*) of apricots, peaches and cherries. Localized action of strong bluestone solution, free arsenic or other toxic chemicals on peach leaves will cause more or less perforation. The behavior of the leaves of pome fruits —apple, pear and quince, in which perforation is rare—is in marked contrast to the behavior of the stone fruits. Sometimes extreme cases of

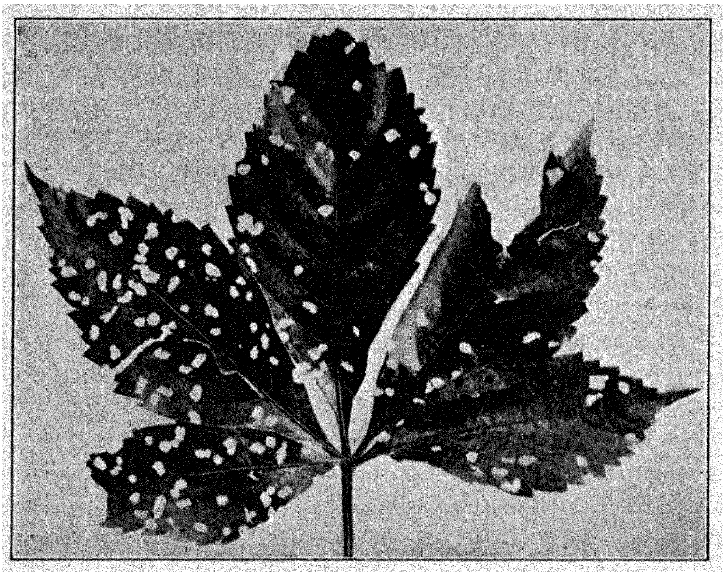


FIG. 13.—Shot hole of Virginia-creeper leaf.

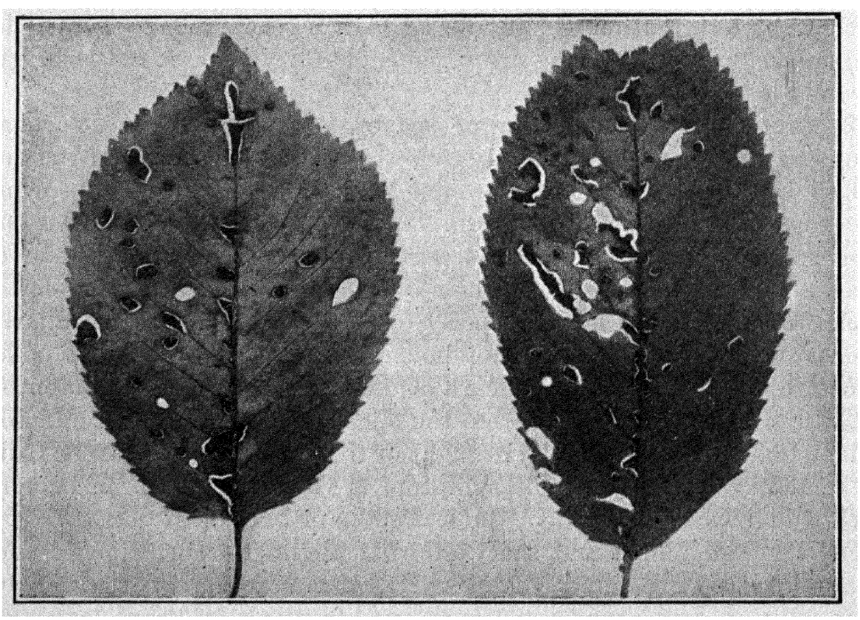


FIG. 14.—Shot hole of cherry.

shot hole may resemble insect work so closely as to be confusing (Cercospora and Phyllosticta spots of the Virginia creeper). Sometimes the leaves fall before the perforations are complete, while in other cases severely affected leaves will remain hanging until the shot holing has run its course.

3. Wilting.—Two types of wilting may be recognized: the sudden wilting of seedlings, or "damping-off"; and the wilting of growing or adult plants. The damping-off of seedlings is most frequent and severe in crops grown under glass, also in garden crops or in seedling trees in the

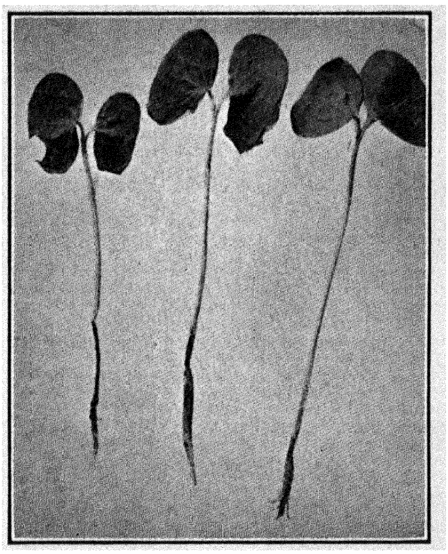


FIG. 15.—"Damping-off" or "sore shin" of cotton due to Rhizoctonia.

nursery, but is less injurious in most field crops. In these cases, the young plant droops over and lies prostrate on the ground or "drops dead," as it were, for a parasite, generally a fungus, has entered the stem at about the ground level and has struck a vital blow, interrupting essential physiological functions. The rigid stem becomes flaccid, and the flow of water from root to seed leaves is interrupted. Recovery is impossible, for the tissues have been killed at about the ground level by the parasite which has entered the young stem from the soil. Various soil-inhabiting fungi may cause damping-off, the most prominent being *Rhizoctonia spp.*, *Pythium debaryanum*, *Thielavia basicola*, *Sclerotinia spp.* and *Sclerotium spp.* Damping-off by killing numerous seedlings in sugar-beet fields is frequently the explanation for poor stands. A damping-off disease of cotton seedlings is prevalent throughout the South and is known to the planters as "sore shin." The rotting of herbaceous cuttings at the ground line in greenhouse benches is generally due to some of the damping-off fungi.

The normal or physiological wilting of growing plants, followed by recovery, should not be confused with the wilting of disease. During periods of bright sunshine in the heat of the day, succulent shoots droop, and leaves may become limp or roll up, due to excess of water evaporation over root absorption, but when lowered temperatures and darkness check

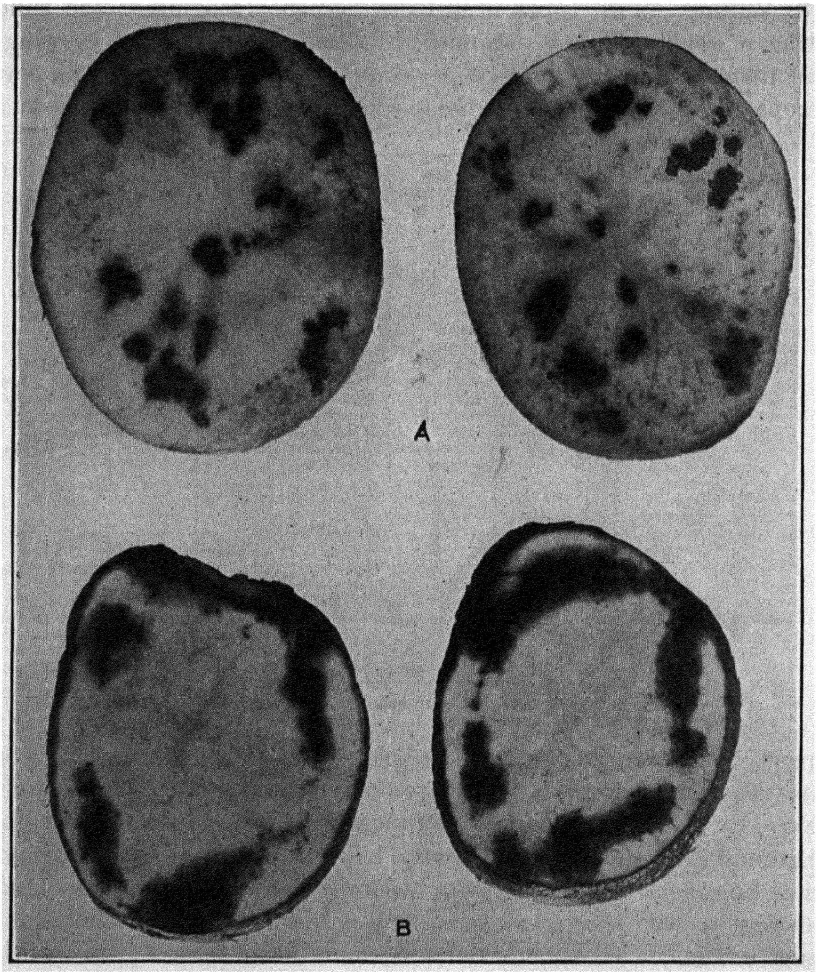


FIG. 16.—*A*, internal brown spot of potato; *B*, bundle browning of potato.

evaporation the flaccid parts again become turgid, and growth continues. In certain cases, the leaves of plants wilt rather suddenly, sometimes with slight premonitory symptoms, and recovery does not follow. Notable cases are the so-called "wilt diseases" of herbaceous plants or the thromboses of woody plants, in which the water-conducting vessels of the hosts are plugged up with either bacteria or fungous mycelium, so that the transpiration current is completely interrupted. The wilt (*Bacillus tracheiphilus*) of cucurbits, the wilt (*B. solanacearum*) of Solanaceæ or

species of the nightshade family, the Fusarium wilts of various hosts and the thrombosis (*Verticillium*) of the maple are typical illustrations.

4. Necrosis, or Death of Parts.—In some diseases, the death of special parts or organs of the plant, as leaves, stems or twigs, buds, flowers or fruits, is the first symptom of disease that is noted, although in many cases wilting is an antecedent phenomenon. The affected structures generally assume the characteristic dark or brown colorations of dead tissue, and the accompanying disease is frequently characterized as a *blight*. This is well illustrated in the fire blight of the apple and pear, in which leaf blight, body blight, twig blight, blossom blight and fruit blight are but phases of a single disease. Necrosis may be localized in certain organs, or it may be more general and result in the death of the entire plant (*e.g.*, severe attacks of late blight, *Phytophthora infestans* of potato).

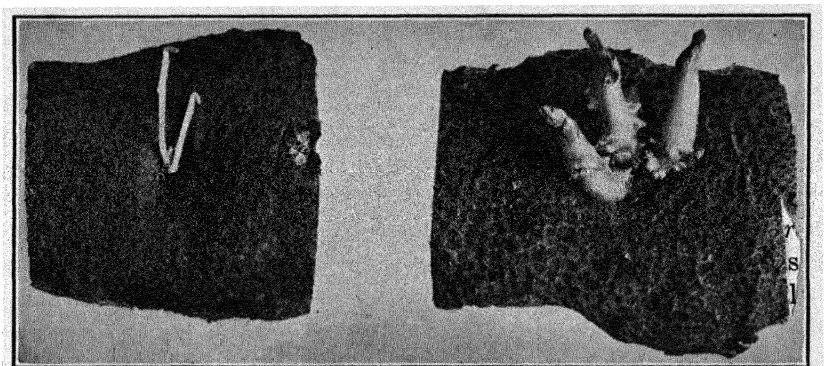


FIG. 17.—Spindling and normal sprouts of potato. (*Photograph by B. F. Dana.*)

Localized areas or groups of cells in certain organs may die, with the result that small or extensive masses of brown, frequently collapsed cells may be found in otherwise normal structures. Internal necroses are well illustrated in the bitter pit or Baldwin spot of the apples and in the internal brown spot, net necrosis and blackheart of the potato, all of which are non-parasitic troubles.

5. Dwarfing or Atrophy.—Either the entire individual or special organs may be reduced in size as a result of unfavorable factors, either environmental or parasitic. Early attacks of parasites which are general upon the foliage, like mildews, rusts and other fungi; diseases which are systemic, like some of the seed-borne smuts (*Tilletia tritici*); or the curly dwarf of the potato may cause the affected plants to remain stunted, or a localized disease like crown gall (*Pseudomonas tumefaciens*) of the apple may cause a permanent retardation of growth. Leaves may be atrophied or reduced in size as the result of localized parasites (some rusts), in witches' brooms; in trees suffering from mushroom root rot (*Armillaria mellea*), crown or collar rot or from lowered vitality due to winter injury; also from various other nutritive disturbances. Reduction in size some-

times suggests the common name of a trouble, as in the little-leaf disease of the apple. Tubers may be reduced in size in the Rhizoctonia disease and in the various degeneration or virus diseases of the potato, and there may be similar results in any root crop. The small size of the fruits is such a characteristic feature that it has suggested the name "little peach," which is applied to a serious contagious disease of that fruit tree, while severely rusted cereals produce shriveled and shrunken grain. It is necessary only to compare normal and diseased plants to note numerous but less striking examples of reduction in size.

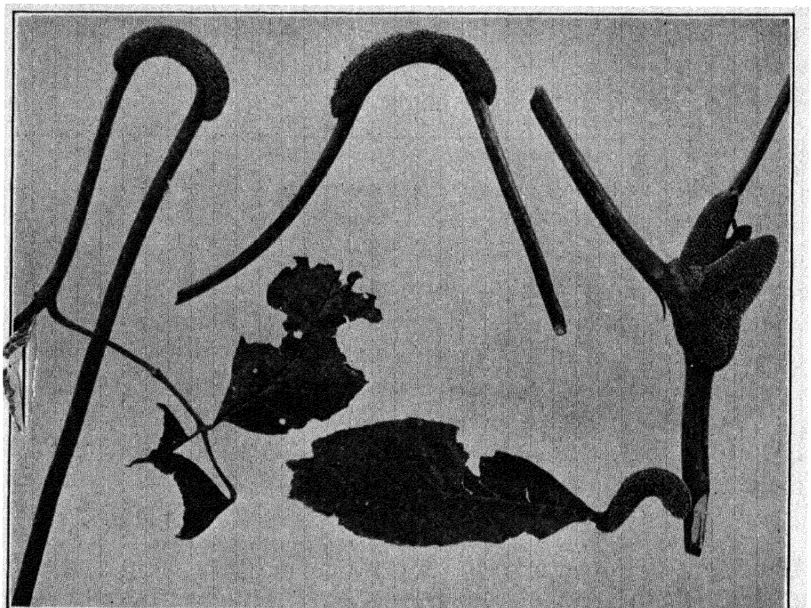


FIG. 18.—Æcial or cluster-cup stage of ash rust (*Puccinia fraxinata*).

6. Increase in Size or Hypertrophy.—Practically all plant organs may be stimulated by the action of parasites so that they are increased in size. The result may be shown either, first, by the increase in the size of the component cells or, second, by increased cell division resulting in the abnormal multiplication of cells (hyperplasia). In some cases, increase both in size and in number of cells may occur. (See also item 12 *B* and *C*.) Hypertrophied roots are well illustrated in various mycorhizas, in the club root, or finger-and-toe disease (*Plasmodiophora brassicæ*) of the cabbage and other crucifers and in the root knot or eelworm disease (*Caconema* (*Heterodera*) *radicicola*) of numerous hosts; hypertrophied stems as in white rust (*Albugo candida*) of crucifers, stem invasions of the Vaccinium rust (*Calyptospora columnaris*) and in some Exobasidium diseases of certain species of the heath family; hypertrophied leaves in the well-known leaf curl (*Taphrina deformans*) of the peach, in the rose

bloom (*Exobasidium vaccinii*) of the cranberry, in which the pink enlarged leaves are grouped to resemble a flower, in various rusts and in many other fungi; hypertrophied flowers or flower parts, as in the white rust (*Albugo candida*) of radish, in infections caused by various Exoascales or leaf-curl fungi; and in certain species of Exobasidiales; and hypertrophied fruits, as illustrated by plum pockets or bladder plums (*Taphrina pruni*) and the leaf-curl fungus (*T. farlowii*) of the chokecherry.

7. The Transformation of Organs or the Replacement of Organs by New Structures.—In the ergot of rye and other grasses (*Claviceps spp.*), certain of the ovaries are destroyed by the action of the parasite, and, in the place of the seeds, horny, elongated, dark-purple, spur-like bodies, the sclerotia or "ergots," appear. These are resting structures composed of a dense aggregate of fungous tissue and serve the purpose of carrying the parasite over the winter period. In normal flowers the sepals, petals, stamens and pistil occupy definite positions, but under abnormal conditions one kind of floral organ may be transformed into another kind or into ordinary leaves. Stamens may become leafy in the green-ear or downy-mildew disease (*Sclerospora graminicola*) of *Pennisetum typhoideum* and other grasses; the whole staminate head, or tassel, or the ear becomes a leafy structure in certain phases of the head smut (*Sorosporium reilianum*) of corn; petals may become like sepals, stamens like carpels and carpels leaf-like in the white rust (*Albugo candida*) of crucifers; all the floral parts are transformed into foliage leaves in the Japanese plum affected with rust (*Cœoma makinoi*). The term "phyllody" is applied to the change of floral organs into leafy structures. While phyllody is frequently caused by the presence of a parasite, other disturbances may produce similar effects.

8. Mummification.—The transformation of fruits into shriveled structures called "mummies" is a phase of numerous diseases affecting our commercial fruits and may also occur in the fruits of wild plants. The fruit generally undergoes an initial change which would be characterized as rotting, but during this process the tissues become filled with the mycelium of the parasite, and the dry, shriveled structure which results consists of the remains of host cells mingled with this mycelium alone or also with spore fruits which have been organized. Mummies remain hanging on the tree or fall to the ground but resist further decay until a later time or after they have produced a crop of spores. Spores may develop at once or not until the next spring or in some cases not until the mummies have passed through two winters. In cases where mummies produce spores in the fall, this process is continued into the following spring, so that mummies may be looked upon as devices to provide for the overwintering of those parasites by which they are produced. The formation of mummies is a very characteristic feature of the brown rot (*Sclerotinia spp.*) of stone or pome fruits, like apricots, peaches, plums,

cherries and apples or pears. In plums or peaches, the brown, shriveled mummies, frequently cemented into clusters which remain hanging on the trees, are a familiar sight in regions where the brown rot of these hosts is prevalent. Other typical illustrations may be found in the bitter rot (*Glomerella cingulata*) and black rot (*Physalospora malorum*) of the apple and in the black rot (*Guignardia bidwellii*) of the grape.

9. Alteration in Habit and Symmetry.—Some plants which under normal conditions are more or less prostrate or creeping become ascending or even erect when attacked by a fungous parasite and, as it were, signal their distress. This is notably true in purslane (pusley), a common garden weed (*Portulaca oleracea*), when attacked by white rust (*Albugo portulaceæ*), and in certain species of spurges (*Euphorbia spp.*) harboring the cluster-cup stages of rust. The same symptom is shown in some tree diseases in which normally horizontal limbs or branches become grouped into more or less erect clusters (see Witches' Brooms under Art. 12). In these cases, a dorsiventral symmetry is changed to a more or less evident radial symmetry. Closely related to the above is the change from the rosette to the cauline type in *Launea asplenifolia*, the unbranched stem and radical leaves being changed to a much branched axis with cauline leaves, as the result of a rust (*Puccinia butleri*).

The vegetative organs may show various alterations; *leaves* may be changed from simple to irregularly lobed, as in a South American barberry attacked by a rust (Æcidium). *Sempervivum hirtum* with obovate leaves normally twice as long as broad may be changed to produce leaves seven times as long as broad when infected with a rust-like fungus (*Endophyllum sempervivi*). Leaves may be variously twisted or deformed by various parasitic attacks (numerous rust infections); stems may be twisted or deformed, and internodes elongated or shortened; and branching may be reduced (various rusts) or increased, as in the degeneration disease of the potato known as witches' broom (see also Hairy Root and Witches' Brooms).

The reproductive structures may be transformed by the action of disease: In the club types of wheat, the head is changed to the elongated or "vulgare" type by the presence of bunt or stinking smut (*Tilletia tritici*); in a species of Acacia, the inflorescence is changed from a head to a spike by a rust (*Œcidium esculentum*); flowers may be changed from regular to irregular (actinomorphic to zygomorphic) or *vice versa*, cyclic types to strobilate types, diœcious to perfect, and various other modifications of the flowers or flower parts may result.

10. The Destruction of Organs.—The complete destruction of organs may result either from non-parasitic causes or from the inroads of a pathogene. The rudiments of seeds—the ovules—may dry up without the production of seed, owing to low-temperature injuries, lowered vitality or failure of fertilization, as in the empty or partially filled cells of an

apple, or seeds may be destroyed by the action of a parasite, as in the smut of sheep sorrel (*Oxalis spp.*), in which the spores of the parasite (*Ustilago oxalidis*) are forcibly expelled from the seed capsule just as if true seeds were present. Entire flowers may be destroyed, or only flower parts, as in the anther smut of pinks caused by *U. violacea*. In the case of "bladder plums" due to *Taphrina pruni*, the pit fails to develop and the rudiment of the seed is destroyed, producing the empty inflated "fools" characteristic of this disease.

The so-called "seeds" or fruits of cereals (the caryopses) are frequently destroyed by the operation of a parasite. This is notably true in the covered or kernel smuts—kernel smut (*U. levis*) of oats, bunt or stinking smuts (*Tilletia tritici* and *T. levis*) of wheat and kernel smut (*Sphacelotheca sorghi*) of sorghum. In these kernel smuts, the young mycelium of the fungus enters the young developing ovary and sporulates there, using up the food that would normally be stored in the seed. The final result is the "smut ball" or spore mass enclosed by a remnant of the wall of the kernel. All or part of the grains in a head may be destroyed, and all or only part of the heads on an infected plant may be smutted. In other cases, the destruction may be more complete and the entire inflorescence may be involved. This is well illustrated in the loose smuts of cereals—loose smut (*Ustilago avenæ*) of oats, loose smut (*U. tritici*) of wheat and loose smut (*U. nuda*) of barley. In a typical case, as in the loose smut of wheat, all parts of the head except the central axis, or rachis, are destroyed and replaced by a black powdery mass which has been largely dissipated by harvest time, leaving the rachis naked or with only a few remnants of the spore mass.

11. Dropping of Leaves, Blossoms, Fruits or Twigs.—This is, of course, to be considered as a symptom of disease only when it occurs prematurely or in excessive amount. It may first be illustrated by the normal behavior of leaves, which are all shed in deciduous species at the end of the growing season, or by the loss of a part of the needle leaves each season by coniferous evergreens. Under such conditions, the leaf falls because of certain changes which take place in the *abscission layers*, or cleavage zone, at the base of the petiole, causing a separation. Nonparasitic influences or parasites acting upon the growing plant may bring about the same changes in the abscission layers as the cold of autumn, thus producing premature leaf fall. Blossoms, twigs, fruit spurs or fruits may be separated from their points of attachment in a similar manner.

Owing to a sudden change of external conditions from a moist greenhouse to a dry room, from moderate to intense light or from cool conditions to warm, such house plants as fuchsias, foliage begonias, azaleas, rubber plants and many others will drop their leaves. The dropping of the leaves is such a characteristic symptom of certain diseases as to suggest the common name, as in the leaf casts (*Hypoderma* or *Lophodermium*

spp.) of the pine and larch. The shedding of lower leaves which have become spotted and chlorotic is a very characteristic behavior of alfalfa plants affected with leaf spot (*Pseudopeziza medicaginis*) or yellow leaf blotch (*Pyrenopeziza medicaginis*). In severe attacks, some cherries affected by the yellows or leaf spot (*Coccomyces hiemalis*) may be nearly defoliated by midsummer. The shedding of blossoms may be illustrated in the blossom drop of the tomato, which is frequently very noticeable during the first part of the growing season when cool, cloudy weather prevails; and in the so-called shelling of grape blossoms or of partially developed berries. It is probable that the shelling or dropping of the reproductive structures is the result of insufficient food to supply both vegetative and reproductive activities. In hyacinths given too much water and subjected to high temperatures at the beginning of the growing season, the stalk bearing the entire inflorescence may separate at the base. Dropping of fruits may be illustrated by shedding of bolls in cotton or the June drop of stone or pome fruits, the former following extremes of weather, either dry or wet, while the latter may be induced by various factors affecting the nutrition of the developing fruits. Parasites localized on the fruits or fruit pedicels may cause dropping of young fruits, as in scab (*Venturia inaequalis*) of apples or California blight (*Coryneum beijerinckii*) of the cherry. The casting of fruit spurs may occur in pears or apples, or there may be an abnormal abscission of twigs and small branches in such trees as willows and poplars due to excess of water or to temporary drought followed by an excessive supply of moisture.

12. The Production of Excrescences and Malformations.—A great variety of abnormal formations may be grouped under this heading. Attention may be directed first to a condition known as *erineum*, which is the name applied by early mycologists to an abnormal development of hairs or trichomes from the surface of leaves giving felt-like patches. While these formations were at first supposed to be of fungous origin, they are now known to be due to the effects of certain parasitic mites and are designated as *erinose*. Common instances may be found in the erinose of the vine, in which young trichome felts suggest the white spots of fruiting downy mildew, and in the erinose of the mountain maple with its striking patches of red or scarlet hairs.

Knot-like or pustule-like distensions of tissue, occurring most abundantly on leaves but also on stems and fruits, due to the abnormal elongation of groups of cells, with or without increased cell division, are called *intumescences*. No parasite is concerned in the formation of these structures, which result from environmental factors. In a typical leaf intumescence, groups of palisade parenchyma cells are abnormally elongated, so that a raised, blister-like pustule is formed. In other cases, they may rupture the epidermis of leaf or twig and make a rough, hairy papilla, which may later become brown and collapsed, due to the death of the

cells. Intumescences are not uncommon on the unripe pods of peas. They are in the form of pale, irregular warts, which by their appearance almost suggest a fungous infection, especially if they have been exposed to very moist conditions. Woolly streaks which appear in the core walls of apples under certain conditions are formed by thick bunches of elongated cells or cell rows and are quite similar in origin to external intumescences. If the overdevelopment of cells is somewhat general, rather than distinctly localized, and extensive swellings of organs result, the condition is spoken of as "œdema" or dropsy. This is not uncommon when there has been pronounced stimulation of growth or a lessened consumption of water with unabated absorption. The condition on apple

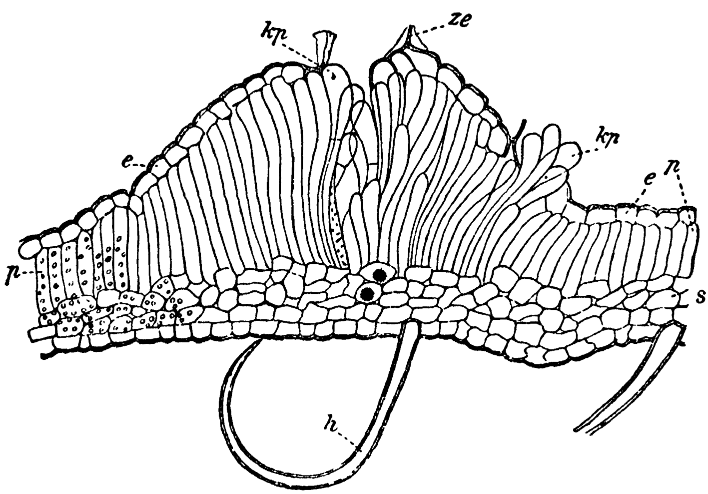


FIG. 19.—Leaf intumescence of *Cassia tomentosa.* (*After Sorauer.*)

branches and roots in which the elongated cells separate and dry to make a fine, brown powder—the so-called "tan disease"—is very similar to œdema.

Localized enlargements on various organs in the form of small pustules or warts, larger tubercles, tumors or masses of cells making a morbid outgrowth of either fleshy or woody character, in which host tissues and parasites mingle, are generally designated as *galls*. These abnormal growths may be due either to plant parasites, mostly fungi or bacteria, or to various insects. The study of galls and their causal agents constitutes a special field of science known as *cecidology*. A few illustrations will be cited to show something of the variety of gall formation by plant parasites: Very small reddish galls, $\frac{1}{25}$ inch in diameter or slightly less, are produced on stems, leaves and other parts of the cranberry in the gall disease (*Synchytrium vaccinii*); the large, rounded or irregular tumors of corn smut (*Ustilago zeæ*) up to 6 inches or more in diameter represent about the extreme in size of galls on herbaceous structures. The so-called

"cedar apples" of our common red cedar, evident as brown, more or less spherical enlargements, 1/8 to 2 inches in diameter and quite fruit-like in appearance, are the result of a rust (*Gymnosporangium juniperi-virginianæ*). The more or less elongated, rough, black outgrowths—the black knots—on the twigs and branches of plums and cherries illustrate a different type of fungous gall (*Plowrightia morbosa*). Bacterial galls (*Pseudomonas tumefaciens*) are well illustrated in the woody or fleshy outgrowths from the crown of fruit trees and numerous other herbaceous

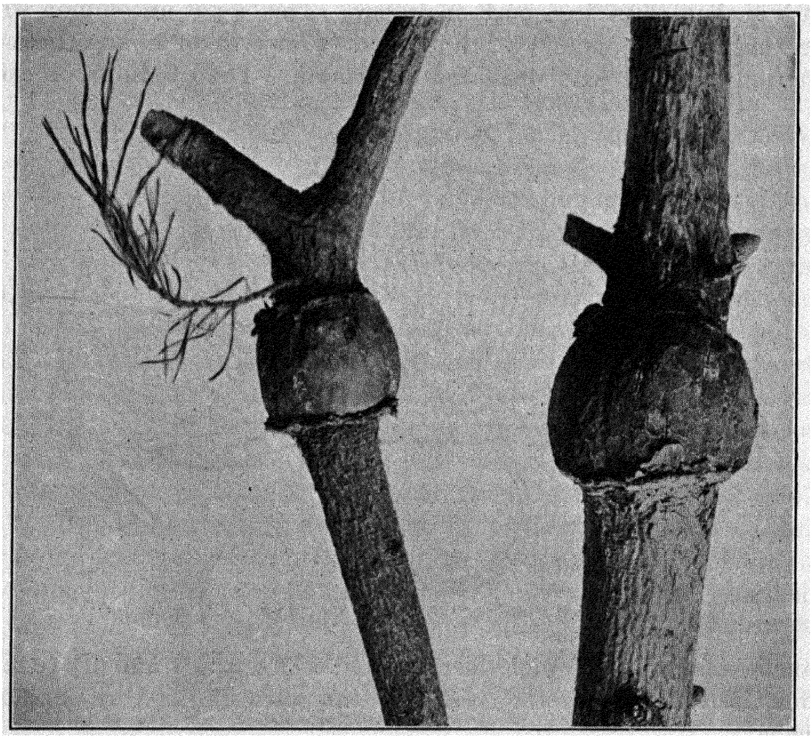


Fig. 20.—Peridermium galls on *Pinus virginiana*.

or woody plants, hence the name *crown gall*. While the graft union is a very common place for such galls, they may appear on any part of the root system and also upon aerial parts, as, for example, on the blackberry, where they involve long stretches on the canes and are sometimes referred to as cane galls. The galls on the leaves, twigs, branches or trunk of the olive, very small on the leaves but varying in size from that of a garden pea to others 2 to 3 centimeters in diameter on the branches or trunk, are also of bacterial origin (*Pseudomonas savastanoi*). The club root or finger-and-toe disease of cabbage, turnips and other species of the mustard family, characterized by enlarged roots or tumor-like outgrowths from the main root, is the evident feature in one of the important chytrid diseases (*Plasmodiophora brassicæ*).

Localized lesions on woody or more rarely herbaceous stems which generally result in the corrosion and sloughing away of tissue with the final production of an open wound, exposing or penetrating the wood, are designated as *cankers*. Young cankers are at first evident as localized discolored areas upon the bark, but with the death of the tissue involved there are shrinking, cracking and shredding and the lesion comes to be more pronounced. A fungous canker may advance slowly or rapidly and may reach its full size in the course of a single growing season (infection in fall of previous year), as in the black spot or Pacific Coast canker (*Neofabrœa malicorticis*) of the apple, or it may be perennial, spreading in an ever widening zone from year to year till the infected branch or trunk is girdled and killed as in the Endothia canker (*Endothia parasitica*) of the chestnut. In a slow-growing, perennial canker, the host will make efforts to heal the wound or to head off the progress of the parasite by the development of callus around the advancing border of the lesion. In time, the building up of these ridges of callus will be evident as more or less regular or broken concentric ridges which mark either years or periods of growth, as is well illustrated in the European canker (*Nectria galligena*) of the apple or in the Strumella disease (*Strumella corynoidea*) of oaks and chestnuts. The majority of canker-producing fungi develop mainly in the bark or with a very limited penetration of the wood, although in other cases (the last mentioned) the parasite may eat into the wood and cut the trunk in two or so weaken it that it is unable to withstand the force of strong winds. Cankers of bacterial origin are well illustrated in the canker stage of the fire blight (*Bacillus amylovorus*) of the apple, pear and other hosts. The bacteria in the majority of cankers die during the first season, but they survive the winter in a limited number, which are then called "holdover cankers." While the majority of cankers are of fungous or bacterial origin, they may be caused by non-parasitic influences, as is well illustrated in the "winter-sun-scald" cankers which appear on the

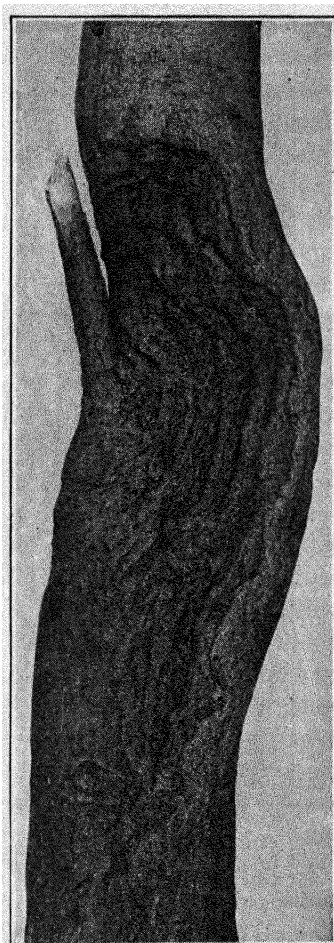


FIG. 21.—Perennial canker of chestnut oak due to *Strumella coryneoidea*.

southwest side of tree trunks, on the exposed faces of larger limbs or in the crotches of the larger limbs.

Various woody plants may produce closely grouped clusters of fine, slender branches, generally arranged more or less parallel to each other

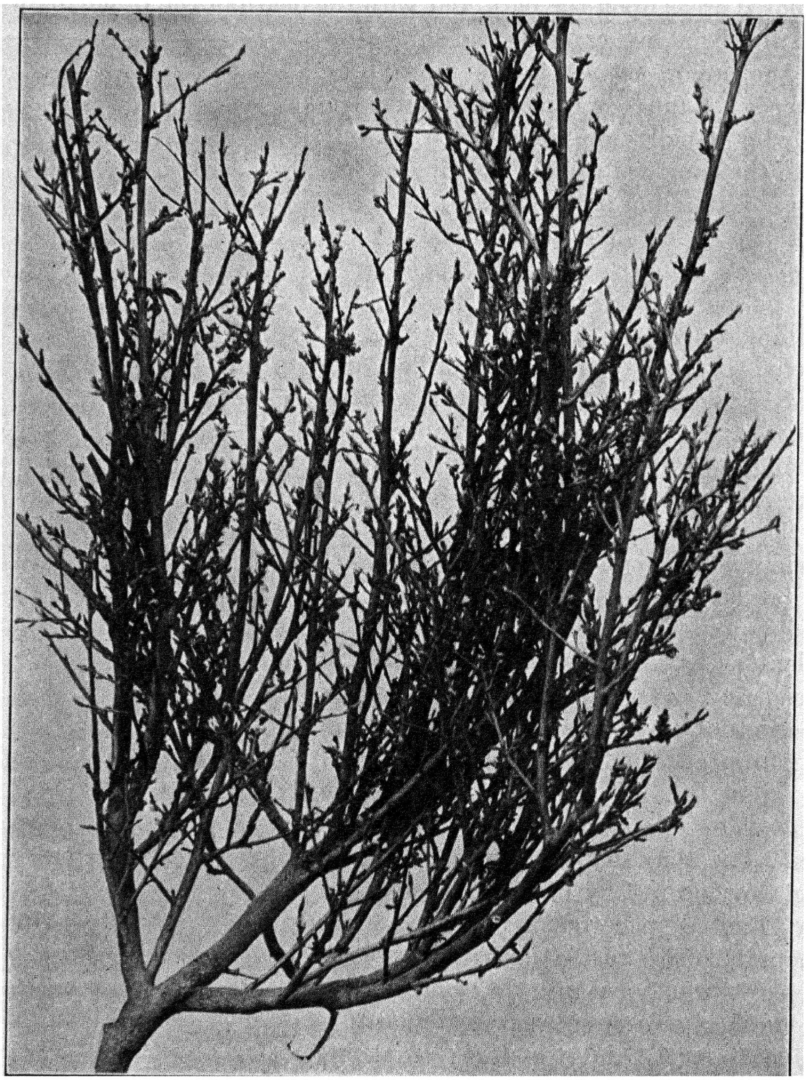


FIG. 22.—Witches' broom of service berry due to *Apiosporina collinsii.*

and frequently originating from an enlarged axis. These broom-like clusters are called "witches' brooms," which is a literal translation of the German name "Hexenbesen," originally applied to these structures by the superstitious peasants of central Europe, who attributed their origin to the machinations of witches. In the majority of cases, the

branches that normally would be horizontal, or only slightly ascending, become erect or nearly so. Various leaf-curl fungi which become perennial in the host tissue are responsible for witches' brooms, as is well illustrated by the leaf curl and witches' broom (*Taphrina cerasi*) of the cherry; rust of various species may give rise to very characteristic brooms on various conifers (*e.g.*, *Gymnosporangium libocedri* on the incense cedar); *Melampsorella elatina* on the silver fir; while the scaly or dwarf mistletoes (*Razoumofskya spp.*) may be the inciting agents in pine, larch and fir trees. The majority of witches' brooms are due to plant parasites, but in some cases insects may play a part, as in the branch knot of the hackberry (*Celtis occidentalis*), caused by a mite (Phytoptus) followed by a fungus.

A development somewhat similar to the witches' brooms of aerial structures may occur on the root systems of woody plants, giving rise to an abnormal number of fine fibrous roots, frequently making compact clusters, the so-called "hairy root." These roots may originate from the main root or the crown, from some lateral root, or they may come from the surface of a crown gall, giving what is known as the woolly-knot form of hairy root. The primordia of adventitious roots may sometimes be organized in closely aggregated groups on trunk or branches but, failing to develop into true roots, may produce rough, papillate, gall-like formations—the aerial hairy root of apple, pear or quince.

Just as some plants which normally have the rosette habit give up this type of growth as a result of disease, others which normally never produce rosettes adopt the rosette habit when they are diseased. In the formation of these rosettes, axes which normally are much elongated remain short and congested due to failure of the internodes to elongate, and as a result the leaves are closely grouped. This rosette symptom is so characteristic in certain diseases that it has given rise to the common name of the trouble, as in the apple rosette of the Pacific Northwest, the pecan rosette of the southern United States and the contagious rosette disease of peaches and plums of the southeastern states. Somewhat similar modifications of growth may accompany diseases of herbaceous plants.

Disease may cause the development of dormant or rudimentary structures, or entirely new organs may appear. Dormant buds may be started into growth, producing new shoots, as may be noted on ringing or girdling diseases of trees, in which the new development is just below the zone of attack, as in silver leaf (*Stereum purpureum*) of fruit trees. Stamens that normally are rudimentary in the pistillate flowers of *Lychnis dioica* when attacked by anther smut (*Ustilago violacea*) grow to full size but produce smut spores instead of pollen; or buffalo grass (*Buchloë dactyloides*) may form ovaries in its staminate flowers when parasitized by smut (*Tilletia buchlœana*). The formation of new organs or structures, either similar to or entirely unlike any normal parts of the host, may occur on leaves, stems or flowers. Striking illustrations are found in the

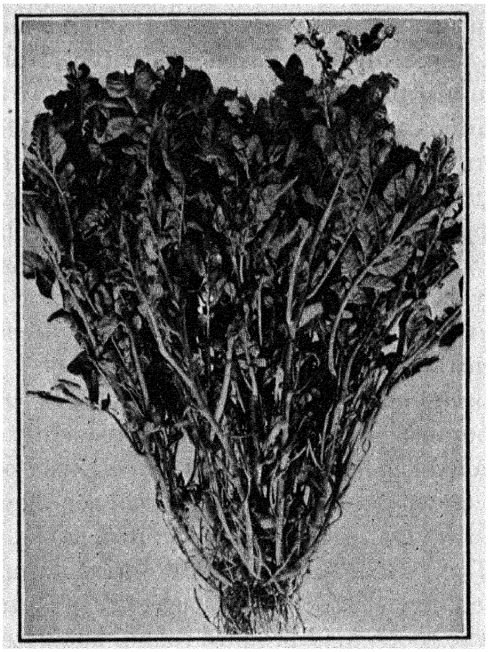


FIG. 23.—Witches' broom of potato. (*Photograph by B. F. Dana.*)

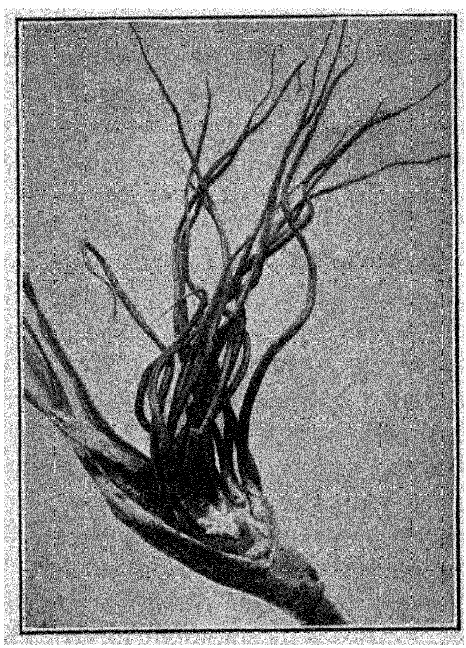


FIG. 24.—An effect of head smut (*Sorosporium reilianum*) on ear of corn. (*Photograph by B. F. Dana.*)

production of slender, branched, leaf-like outgrowths from the fronds of a fern (Pteris) infected with one of the leaf-curl fungi (*Taphrina laurencia*); or in the pallid, leafless branched structures which appear on shoots and leaves of a Japanese conifer (Thujopsis), when infected with a rust (*Cœoma deformans*). New flower parts may appear, as in *Viola silvestris*, which produces extra petals when affected with rust.

In certain cases, an organ may continue to produce new growths after it has reached the form or stage which would normally end its develop-

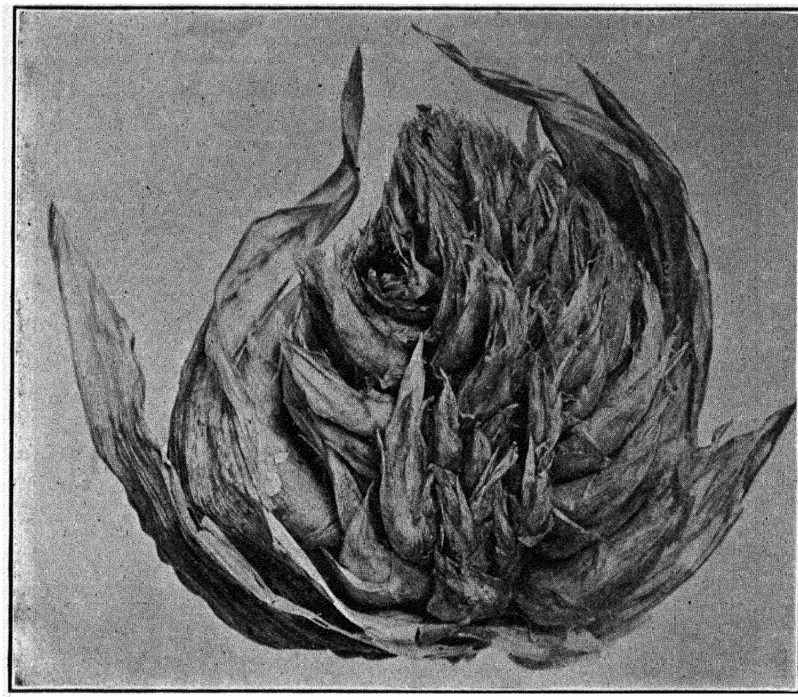


FIG. 25.—An effect of head smut (*S. reilianum*) on ear of corn. (*Photograph by B. F. Dana.*)

ment. This symptom—*proliferation* or *prolification*—is well illustrated in the conifers, especially the larch, in which the cone axis may grow out into an elongated, needle-bearing branch; in roses, in which a new blossom may sometimes come from the center of an old one; in certain composites, in which peripheral florets may grow into elongated axes bearing small but perfect flower heads; in the so-called "sprouting pears" (also other pome fruits), in which one pear may be formed directly above another or a leafy twig may continue to grow from the calyx end; or in the second growth or sprouting of potato tubers while still in the soil. In such cases, there is a disturbed nutrition giving an oversupply of plastic food substances. Somewhat similar disturbances may result from the attacks of fungi, as in the "green-ear disease" or downy mildew (*Sclerospora graminicola*) of certain grasses in which the head is transformed wholly or

in part into a loose green head, due to the growth of florets into small, leafy shoots; or in corn affected with head smut (*Sorosporium reilianum*), in which flowers or an infected inflorescence sometimes grows out into either leafy structures or small leafy shoots with the elimination of spore production.

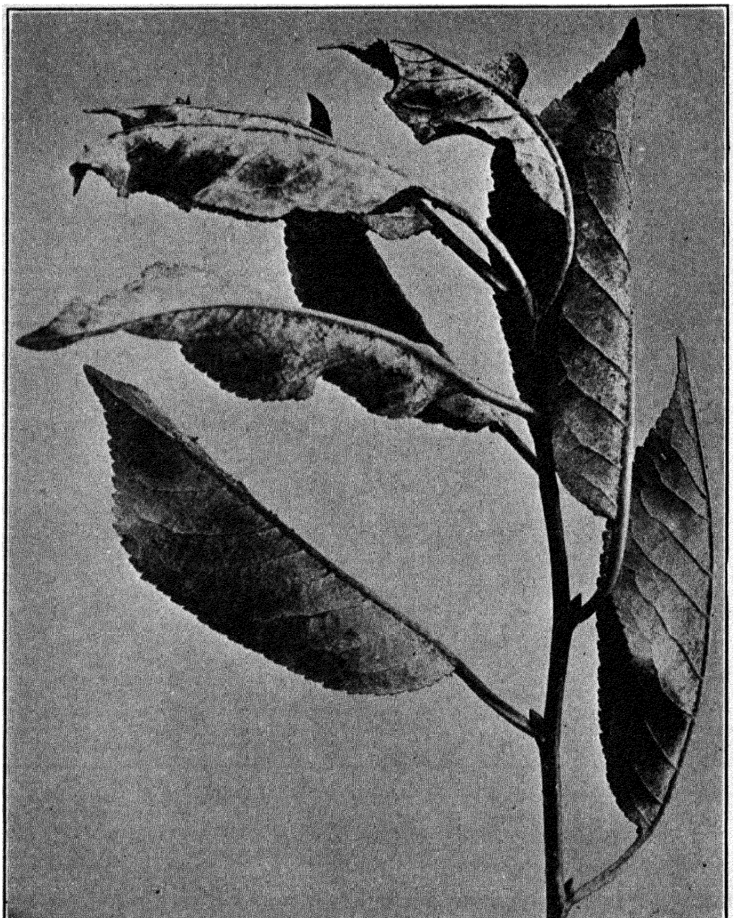


FIG. 26.—Rolling of cherry leaves due to powdery mildew (*Podosphæra oxyacanthæ*).

Rolling, crinkling or curling of leaves may result from the operation of either parasitic or non-parasitic factors. Rolling of leaves is the first signal of thirst in many plants and may serve as a temporary check to loss by evaporation. This symptom is so characteristic in certain troubles that it has suggested the common name, as in the leaf curl (*Taphrina* (*Exoascus*) *deformans*) of the peach; or the leaf roll and curly dwarf of the potato, belonging to the degeneration or virus diseases. In other cases, diseased leaves, instead of showing the normal smooth surface, may be

variously crinkled or puckered, as in numerous mosaic diseases or in apple leaves showing frost blistering.

Cylindrical organs may become broad, flattened and more or less band-like, as though a number of branches had grown together. Such banded or *fasciated* structures may be more or less twisted or thrown into coils or spirals. The entire inflorescence of some plants may undergo general fasciation, as has occurred in the cockscomb (*Celosia cristata*), in which the abnormality has been propagated for ornamental purposes.

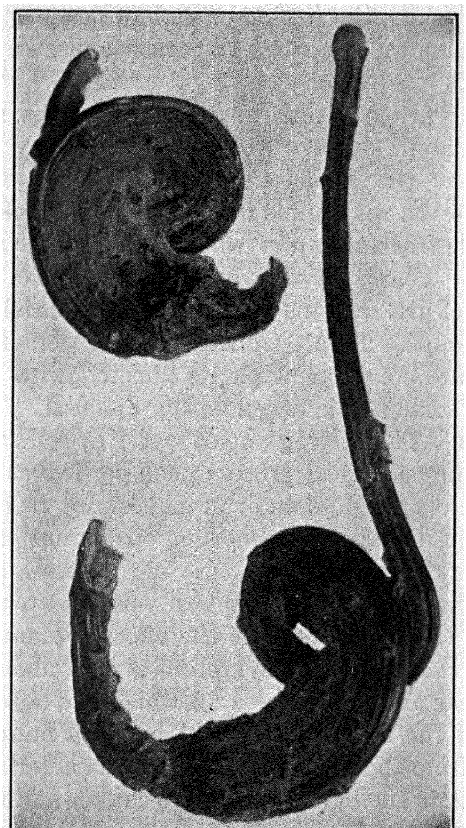


FIG. 27.—Fasciation of apple twigs.

Cylindrical stems may show little or even no evident flattening but may grow in such a manner as to produce an irregular scraggly growth with a flat or open spiral form, or the spiral form may be noted in the *spiralism* of certain woody plants (*e.g.*, Yellow Newtown apple or the Gravenstein, where it has come to be almost a variety characteristic).

The abnormal roughening of surfaces, which under normal conditions should remain smooth, is another symptom of disease. This roughening may be very slight, as in the russeting of the skin of apples or pears, which may be due to frost injury, to use of a spray like Bordeaux or to the

attacks of a parasite as in the net russeting of apples from powdery mildew (*Podosphæra leucotricha*). Russeting may be general or diffuse, or it may be localized in the form of small patches, rings or bands. In some varieties, russeting in part or throughout has come to be a normal variety character. If the roughening of the surface is more pronounced, the name "scurf" or "scab" is frequently applied to the condition, as may be illustrated by the rough-bark disease of the apple and pear (non-parasitic), the rough-bark canker (*Phyllosticta prunicola*), the scab of citrus fruits, the scab of apples and pears, the freckle or scab of peaches and the common scab of potatoes, each an infectious or parasitic disease due to a specific pathogene.

Fruits may be variously deformed, or root crops may be cracked, corroded or irregular in shape. Deformed fruits may result from such parasitic attacks as the apple scab fungus (*Venturia inæqualis*), or sucking insects like the rosy aphis may produce irregular, gnarly apples (Stigmonose), while freezing injury when the fruits are young may cause other deformities. Pears may develop an excessive stoniness, or an increased number of grit cells may be formed (Lithiasis), either scattered or in localized groups. Potato tubers that are cracked, corroded or irregular in form are common in plants affected with Rhizoctonia and also in the case of some of the degeneration diseases.

13. Exudations.—The forcing out of water or cell sap upon free surfaces is a normal physiological process in many leaves (guttation), and *bleeding* from the cut ends of stem or branches may be expected in many cases, but in certain diseases the abnormal oozing out of watery or slimy products which may or may not set into hard masses is a very striking symptom. The character of the exudate varies with the nature of the causal factors and the peculiarities of the affected plants.

A. Bacterial Exudates.—This symptom is well illustrated in the fire blight of apple, pear and other hosts, in which the ooze or exudate consists of myriads of the minute fire-blight bacteria mingled with decomposition products from the affected tissue. The bacterial ooze may burst out through a break in the bark of an active canker and run down the side of the trunk or branch. At first, or under conditions of abundant moisture, it is a sticky, fluid mass, but with evaporation of moisture it may set into firm masses which remain until washed away by rains. On succulent structures like 1-year-old twigs, leaf stalks, flower pedicels or fruits, the ooze may solidify into minute, pearl-like droplets, which are clear or slightly tinged with yellow. In various other parenchyma types of bacterial diseases, exudate will be formed, even in bacterial leaf spots spreading as a thin film or crust over the surface of a lesion.

B. Slime Flux.—This name is applied to the fluid or semifluid outflow, from the bark or wood of various deciduous trees, which does not set into solid masses. Wounds may bleed, and the exudate, which is really a good

nutrient solution, may support various bacteria or fungi which delay or prevent the healing process, while in other cases decomposition products of the diseased tissue may be formed and finally poured out upon free surfaces, sometimes with the development of pockets due to the breaking down of affected cells. Parasitic organisms may be associated with slime flux, but in other cases they are to be viewed as accompaniments of tissue derangement.

C. Gummosis.—This term is applied to the formation of clear or amber-colored exudates which set into solid masses upon the surface of affected parts. Internal gum pockets may be formed which become filled with the decomposition products of the diseased tissue, or the decomposition products may be forced out upon some free surface where they accumulate. With continued disintegration of tissue, the gum masses may increase in size, since they are not soluble in water. Gummosis is a peculiarity of certain plants and is therefore no reliable indicator of any specific parasitic or non-parasitic disturbance. It is an accompaniment of many diseased conditions in stone fruits, especially in cherries and peaches, and is also common in citrus stock. In the above-mentioned plants, gummosis is likely to follow a localized death of cells from any cause whatever. In the cherry, for example, it may result from growth in heavy, poorly aerated soils, from too deep setting, from winter injury, from excessive pruning, from the presence of pathogenic bacteria—*e.g.*, bacterial gummosis (*Pseudomonas cerasus*)—from a definite fungous disease—*e.g.*, California blight (*Coryneum beijerinckii*)—or from the injurious effects of an insect pest—*e.g.*, San José scale. It should then be emphasized that gummosis, in itself, is not a disease but a symptom of disease which may be brought about in a great variety of ways.

D. Resinosis.—The production of resin by special secretory canals is a normal feature in pines, firs, spruces and other coniferous trees, and the limited occurrence of pitch as a surface exudate is frequent, but the excessive outflow of resin may indicate the presence of disease. Resinosis is an attendant symptom in certain coniferous rusts (*Peridermium spp.*) and may also result from the attacks of wood-destroying fungi, the abnormal production of resin sometimes giving the first signal of the presence of an internal parasite.

E. Latexosis.—The production of latex, or a milky fluid, in special lacticiferous vessels is a characteristic of certain species of plants, and this milky fluid will ooze out from cut surfaces. An abnormal outflow of latex may accompany certain diseases—*e.g.*, the older stages of rubber canker.

14. Rotting.—Succulent or woody stems and roots, fleshy leaves, flower buds or fruits may be affected with either dry or soft rot—the "gangrene" of plant tissue. The character of the rot may depend on the structures involved, the causal factors or complications and external conditions. In the majority of cases, soft or pulpy fruits undergo a soft, or

wet, rot, because of the abundance of water in their tissues, while firmer structures are affected with dry rot unless the initial changes are followed by the attacks of putrefactive bacteria.

a. Root Rots.—Either fleshy or woody roots may be attacked by bacteria or fungi which may cause a rapid or a slow disintegration of the invaded tissues. This effect is well illustrated in the destructive Texas root rot (*Ozonium omnivorum*) and the root rots of alfalfa due to various fungi (*Fusarium spp., Rhizoctonia spp.*). Root crops like beets, carrots, turnips, parsnips, sweet potato, etc., may develop soft rots due to bacteria (*Bacillus carotovorus* and others), or either dry or soft rots may be

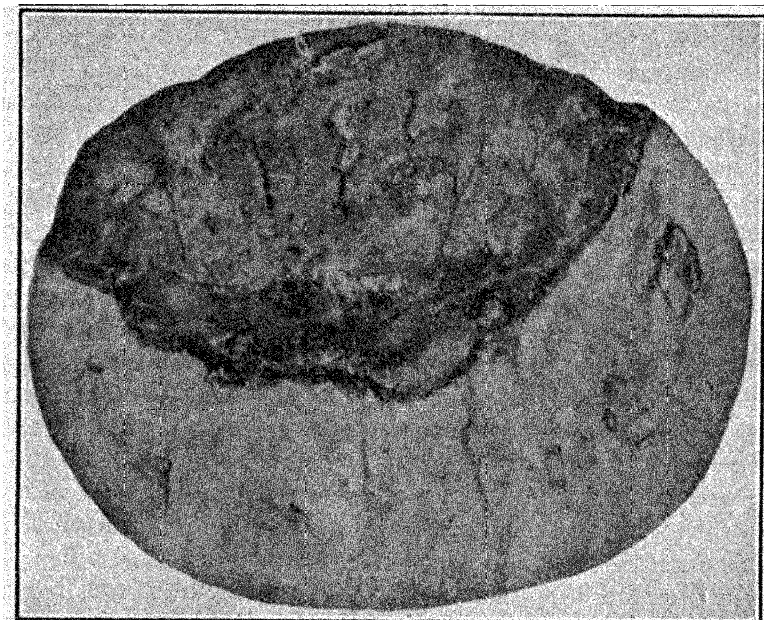


FIG. 28.—Cross-section of the fruit of an eggplant rotted by *Phomopsis vexans*.

caused by fungi (*e.g., Phoma betæ* on beet, Rhizopus rot of sweet potato). Woody roots of our forest and shade trees, or of bush fruits or ornamental shrubs, may be rotted by fungi. The shoe-string fungus or honey agaric (*Armillaria mellea*) as the cause of mushroom root rot is a parasite of wide distribution; the Ozonium of cotton attacks many other hosts, both herbaceous and woody; and various wood-destroying fungi may work in the roots and trunk of affected trees.

b. Leaf or Stem Rots.—Succulent or *herbaceous* leaves or stems may rot from the inroads of either bacteria or fungi, as in the late blight (*Phytophthora infestans*) of the potato, the slimy soft rot of lettuce (bacterial), the stem rot or wilt (*Sclerotinia sclerotiorum*) of various garden and field crops, the stem rot (*Fusarium sp.*) of the cultivated aster, the blackleg (*Bacillus atrosepticus*) of potato and numerous other illustrations. *Modi-*

fied stems, or tubers, rhizomes, bulbs and corms, are storehouses of reserve food and as such furnish an excellent pabulum for bacteria or fungi, and since their tissues are in a dormant or languid condition they frequently fall an easy prey to weak parasites. The potato with its soft and dry rots due to various bacteria and fungi will present typical illustrations: dry rots due to the late-blight fungus (*Phytophthora infestans*) and various species of Fusarium; soft rots due to bacteria following late-blight attacks, to the tuber phase of blackleg or to the inroads of a soil fungus (*Pythium debaryanum*), which produces the disease known as watery soft rot, melters or leak. *Woody stems* of living hosts—standing dead timber or posts, poles and lumber—may undergo decay or disintegration by the action of wood-destroying fungi which grow through or between the wood cells. In living hosts, these fungi are generally wound parasites, entering through some opening which has exposed the wood. Once on the inside, they may continue to grow, sapping the life of the tree or destroying its mechanical support. Certain fungi grow primarily in the heartwood (*heart rots*), while others grow primarily in the sapwood and bark (*sap rots*). Wood that is invaded by fungi may be discolored or made brittle, soft or punky, the exact effects varying with host and fungus. The appearance of fruiting bodies on the bark of trunk or branches or at the crown of a tree in the form of brackets ("punks or conchs" of lumbermen), flat or prostrate, smooth or toothed structures or toadstool-like forms is a signal that the branch or trunk is already pervaded by the vegetative body of the fungus, which is now emerging from its hiding place in order that it may send its offspring in the form of spores to conquer new worlds. Many wood-destroying fungi are pure saprophytes and in such a rôle cause the disintegration of posts, poles and structural timber whenever it comes in contact with the ground or is sufficiently moist to support fungous growth.

c. Bud Rots.—Fleshy buds sometimes offer a congenial home for rot-producing organisms. This may be seen in the bud rot of the carnation, a disease in which the unopened buds may have the enclosed petals and other flower parts rotted by the combined action of a fungus (*Sporotrichum anthophilum*) and a mite. The spores of the pathogene are apparently carried by the young mites which migrate to unopened buds and soon find the soft, rotted petals a suitable substratum in which to complete their development. A second illustration is afforded by the bud rot of the coconut, produced by a fungus (*Phytophthora faberi*) with secondary invasions by bacteria. It is claimed that this disease is spread by vultures which are attracted by the odor of putrefaction. The cabbage, which is really a specialized bud, may be entirely destroyed by the bacteria of black rot (*Pseudomonas campestris*) and secondary invaders which work down the leaf veins and get into the center of the head, finally producing a putrid, foul-smelling mass.

d. Fruit Rots.—The rotting of fruit may result in a few cases from the operation of non-parasitic factors, but the great majority of cases of fruit rot are due to either bacteria or fungi. The best example of a non-parasitic fruit rot is illustrated by the blossom-end rot of the tomato, which generally begins on immature fruits and produces a dry, black rot on the blossom end. The rotted tissue is frequently invaded by fungi, which were at first thought to bear a causal relation to the disease; but it is now known that these are entirely secondary factors and that the decomposition of the tissue is the result of a deranged nutrition. A somewhat similar blossom-end rot of the watermelon has been observed in the South.

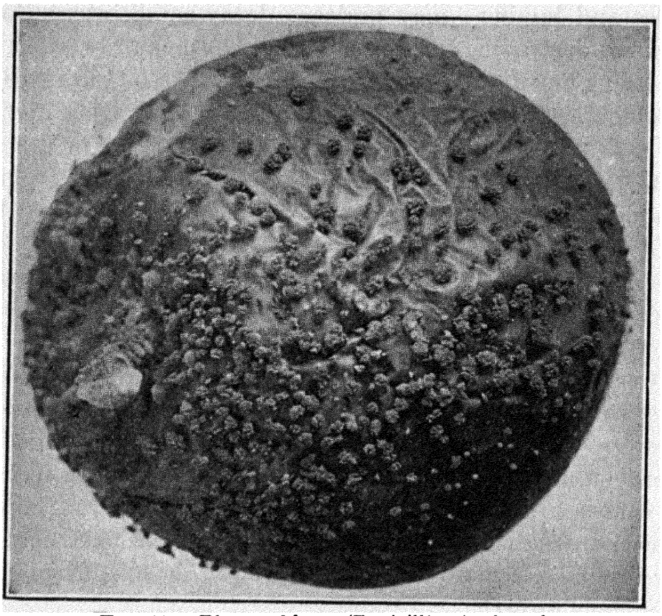


FIG. 29.—Blue-mold rot (Penicillium) of apple.

The great majority of fruit rots of parasitic origin are due to fungi, rather than to bacteria, since the chemical reaction of most fruit juices seems to be unfavorable to the growth of bacteria but favorable to the growth of many different fungi. The soft rot (*Bacillus melonis*) of the muskmelon is due to bacteria which gain an entrance through some wound and cause a complete decay with an offensive odor. Bacteria of fire blight will grow in young fruits of susceptible hosts but are never responsible for the rotting of mature fruit or fruit in storage. None of the transportation or storage rots of small fruits, stone fruits or pome fruits is caused by bacteria, but numerous fungi belonging to widely separated groups are very destructive.

The fruit-rotting fungi may gain an entrance through the unbroken skin in numerous cases—*e.g.*, bitter rot of apple (*Glomerella cingulata*)—but many fungi can enter only through a wound or bruise. The rotting

may begin when the fruit is still immature and hanging on the tree and continue during transportation to market or during storage. Many fruit-rotting fungi are weak parasites and attack only ripe fruit, just at the time of maturity or after it has been harvested. Soft fruits like strawberries suffer rapid decay from fungi, while fruits like apples with a protective epidermis may be preserved for a long time. If it were possible to prevent the inroads of fungi, the drying of fruits from loss of moisture would be the factor limiting the keeping of fresh fruit in many cases, although it must be remembered that the cells of fruits are still alive and that chemical breakdown will follow in due time even though moisture is retained and fungi are excluded (*e.g.*, internal breakdown of the apple).

Rots of fruits may be soft or dry, varying with the nature of the fruit, the identity of the causal organism and the environmental factors which are operative. Strawberries attacked by *Rhizopus nigricans* undergo a very soft watery rot which is called "leak," while fruits affected with Botrytis remain firmer and finally dry to shriveled mummies. Apples rotted by blue mold (Penicillum) are soft and decay completely, while attacks of brown rot (Sclerotinia) may finally produce slightly shrunken, coal-black mummies which resist final decay. Fruits which are not fleshy may be attacked by fungi and undergo decay or dry rot, as is well shown in the dry rot (*Diplodia zeæ*) of corn, which is very destructive in the corn belt of the United States. The most important fungi causing rots of our perishable fruits are species of the following genera: Pythiacystis, Sclerotinia, Penicillium, Alternaria and Phomopsis on citrus fruits; Glomerella, Physalospora, Neofabræa, Sclerotinia, Penicillium, Botrytis and Alternaria on apples and pears; Sclerotinia on stone fruits; Botrytis, Rhizopus and Penicillium on strawberries and bush fruits; and Guignardia, Plasmopara and Glœosporium on grapes.

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SECTION II

NON-PARASITIC DISEASES

CHAPTER III

DISEASES DUE TO DEFICIENCIES OF FOOD MATERIALS IN THE SOIL

Considering the varying origin and composition of soils and the fact that plants must obtain the large part of their food materials from the soil environment, disturbances of nutrition due to insufficient amounts of some of the essential food materials may be expected under certain natural field conditions.

Chemical Elements Required by Green Plants.—Every green plant requires ten different chemical elements, and these must be obtained either in the form of elements or combined in compounds which are available in the environment in which the plant grows. These ten essential elements are as follows: carbon (C), hydrogen (H), oxygen (O), nitrogen (N), phosphorus (P), sulphur (S), potassium (K), calcium (Ca), magnesium (Mg) and iron (Fe). Most of the carbon and oxygen are obtained from the air as carbon dioxide and free or elemental oxygen and used by the plant in the process of carbohydrate food manufacture or photosynthesis and in respiration. The great volume of elemental nitrogen which forms four-fifths of the atmosphere is available for only a few specialized plants which have the power to fix free nitrogen. Hydrogen is obtained in part from water. The four elements named are volatilized and lost when plant tissue is burned or reduced to ash, but the other essential elements P, S, K, Ca, Mg and Fe, together with a number of other accessory ones, are left behind in the ash. Crop plants, exclusive of the legumes, must get their nitrogen from the soil in combined form, and all crop plants must derive their ash constituents from compounds which are brought into solution in the soil water. Some of the non-essential elements of the ash serve no important function in the life of the plant, while others may be of value because of a stimulating effect or because their incorporation modifies certain plant structures.

The Uses of the Essential Elements.—Carbon, hydrogen and oxygen are combined to form carbohydrates, which are essential foods for crop plants. In addition to these three elements, nitrogen, sulphur and phosphorus enter into the composition of proteins and nucleoproteins which are manufactured by green plants and utilized in the nutritive

process. *Potassium* is essential for healthy growth and accompanies and plays a part in carbohydrate synthesis *Calcium* is necessary for normal leaf development; it exists as calcium pectinate, in the middle lamellæ, which cements adjoining cells and may serve a protective action by combining with oxalic acid to form crystals of calcium oxalate which are insoluble. This would prevent the injurious effects from the accumulation of oxalic acid. *Magnesium,* if not an actual constituent, at least accompanies certain proteins and is contained in chlorophyll. *Iron* in minute amounts is essential for green plants, and its lack prevents chlorophyll formation. When deprived of iron, plants develop pale or chlorotic foliage. Some of the essential elements may serve in the growth of plants in other ways—for example, sulphur appears to have a stimulating effect on certain crops, while calcium appears to help in maintaining a proper soil reaction.

Elements Likely to Be Deficient.—Carbon, hydrogen and oxygen are generally available to the growing plant in sufficient amounts to satisfy the needs. In certain cases, lack of oxygen may cause asphyxiation of roots or play a part in storage troubles of plant products (Chap. VI on Diseases Due to Improper Air Relations). Water shortage probably causes injuries because of interference with other water functions, rather than by depriving the plants of hydrogen furnished. The principal deficiencies of chemical elements are of those supplied to the plant through soil compounds. Those which are most likely to be deficient in certain soils and to limit plant growth or give rise to abnormal or diseased conditions are nitrogen, phosphorus and potassium. Under certain conditions, manganese, sulphur, magnesium, calcium, iron or even such a toxic element as boron may be lacking in the proper amount. The absence of an element or its presence in non-available form will lead to the same results. The shortage of a single one of the essential elements or of two or more in the soil of fields or greenhouses may simply retard or restrict growth and fruit formation if the shortage is not too pronounced; or if the deficiencies are greater, marked pathological conditions may result.

SAND DROWN OF TOBACCO

A deficiency in the supply of magnesium has been shown to cause disease in various plants (Graebner, 1921), but a chlorosis of tobacco may be described as illustrating this type of derangement. This chlorosis of tobacco which has recently been investigated in some detail has been called "sand drown," because of its occurrence on sandy soils which have been leached by heavy rainfall. The disease was first observed in North Carolina in 1912 and has since been studied in that state and in other tobacco sections.

In general, it may be said that with approximately normal rainfall and the use of fertilizers of the grades and quantities that have been most widely employed

in tobacco culture, sand drown is not commonly seen in sufficiently severe form to attract attention or to cause serious loss (Garner *et al.*, 1923).

The disease is, however, fairly frequent in certain sandy soils in seasons of excessive rainfall.

Symptoms and Effects.—The affected plants are marked by a chlorosis which begins with the tips of the lowermost leaves and advances until the whole leaf is involved and in the most severe cases nearly the entire plant. The chlorosis advances from the tip toward the base or from the margin toward the center of the leaf. The discoloration is not complete,

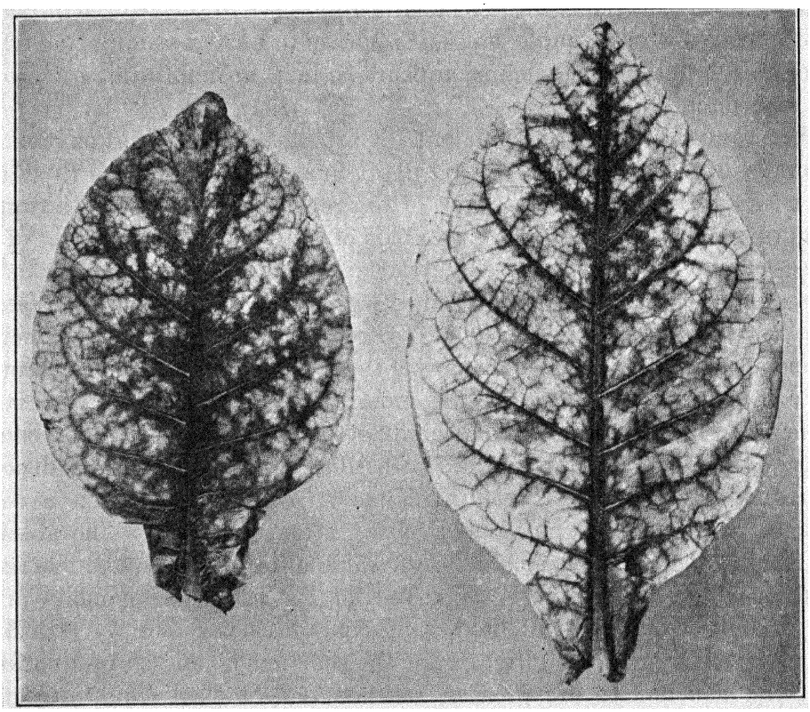

FIG. 30.—Leaves of tobacco affected with sand drown. (*After Garner et al., U. S. Dept. Agr.*)

but the veins and some of the adjacent tissue retain more or less of the normal green color so that affected leaves show a characteristic mottling. The affected tissue becomes a dull, pale yellow or in extreme cases almost pure white, since the green and also the yellow pigments are affected. The color is distinct from the clear lemon or orange-yellow characteristic of etiolated leaves or those which have been blanched by depriving them of light. The leaves of affected plants generally reach normal size, since the disease appears but rarely until the plants are of considerable size. It is more common after the plants have been topped than in earlier stages.

As might be expected in such a disturbance of the chlorophyll apparatus, the cured leaves from affected plants are thinner and have less weight than those from normal plants. In certain tests, normal flue-cured leaves per unit area have been found to be 45 per cent thicker and 65 per cent heavier than diseased leaves. The cured diseased leaves are injured in texture, since they are drier and more lifeless than normal leaves. The color of cured tobacco has an important effect upon its commercial value, and it may be noted that sand drown causes a marked reduction in this respect.

In the flue-cured district normal leaves when properly cured have a bright lemon to orange color, and in other districts the colors range through various shades of brown with either a reddish or a greenish cast. On the other hand, the leaves or the portions of leaves affected with sand drown show, after curing, a dull, faded appearance similar to that observed in the field (Garner *et al.*, 1923).

Sand drown, therefore, may cause a lowering of grade or quality because of impairment of color, reduction in weight and unfavorable effects on the body and elasticity of tobacco leaves.

Tobacco is affected by several other types of chlorosis which should be differentiated from sand drown: (1) potash-hunger chlorosis; (2) pallor due to sulphur deficiency; (3) frenching; and (4) infectious chlorosis or mosaic. The chlorosis of potash hunger is distinguished from sand drown by puckering of the leaves and by a characteristic downward curving of the leaf margins giving the condition known as "rim bound" and by the prompt appearance of small, dead spots in the affected leaves. In sulphur deficiency, the color of the whole leaf is a light shade of green, midrib, veins and intraveinous areas being uniform. In frenching, the leaves are mottled somewhat similar to those of sand drown, but they are smaller and generally narrower than normal leaves. The characteristic mottling of mosaic is distributed uniformly over the leaf and appears only in the growing tissue.

Etiology.—Sand drown has been shown to be due to a magnesium deficiency. According to Garner *et al.* (1923), the disease may result under field conditions when heavy rains remove the magnesium from sand soils by leaching. Also the use of certain fertilizer salts may lead to sand drown even in seasons of normal rainfall and even on the heavier types of soil. It has been possible to produce sand drown in pot cultures by watering the soil with complete nutrient solutions from which magnesium has been omitted. Galvanized-iron buckets with perforated bottoms were used and the nutrient solutions were added in considerable excess so as to cause leaching. By this procedure, sand drown appeared quickly when the magnesium compound was omitted. When the disease had not progressed too far, further proof of the fact that the chlorosis was

due to the deficiency of magnesium was obtained by the ready restoration to normal by the addition of magnesium. It is also stated that:

Under favorable conditions, the disease may be induced by applying the fertilizer salts (with magnesium omitted) to the soil before transplanting and avoiding any leaching action in watering the plants. Naturally, the symptoms are less severe than when leaching is employed.

Sand drown has been greatly increased in severity in field tests by the application of certain fertilizers, while it has been reduced or prevented by others. In fertilizer tests on the Durham sandy loam (North Carolina), it was found that even the smallest applications of potassium sulphate resulted in a marked increase in the amount of the disease and that the chlorosis became more severe with increases in the rate of application of the sulphate. When ammonium sulphate was used as a source of nitrogen, the disease was accentuated, a fact which again demonstrated the injurious action of sulphates.

It has been shown that fertilizing with vegetable forms of organic matter tends to prevent the appearance of sand drown. For example, the application of 500 pounds of cottonseed meal per acre showed a reduction in the disease and delayed the onset of the symptoms. The protecting effects of plant organic matter were shown in fertilizer tests with tobacco stalks and stems and barnyard manure with the addition of the necessary mineral fertilizers, but when dried blood was the source of the nitrogen the symptoms of the disease appeared. The application of common salt at the rate of 200 pounds per acre prevented sand drown, but when the chemically pure product was used no benefit resulted. Sand drown has also been prevented by the use of dolomitic limestone.

It is the belief that the beneficial effect of salt, cottonseed meal, tobacco stems, manure, lime and impure potash fertilizers in preventing sand drown is due to the magnesia supplied by these fertilizers. Common salt contains a small amount of magnesia and is also believed to liberate magnesia when applied to the soil.

Cottonseed meal contains about 1 per cent magnesia, and this offers a possible explanation of the marked preference for this fertilizer material by tobacco growers of the Connecticut Valley where color of the cured leaf is of so much importance. It may be that vegetable material of this character through gradual decay in the soil furnishes a more or less continuous source of magnesia in suitable quantities through the growing season (Garner *et al.*, 1923).

Agricultural lime generally contains more or less magnesium, hence will prevent sand drown, since 20 pounds or less of magnesium per acre is sufficient.

Tests have shown that low-grade potash salts, such as kainit and double-manure salts, eliminate sand drown and produce much better growth than high-grade sulphate. It has been found, in fact, that both muriate and sulphate of

potash in relatively pure form but still containing very small percentages of magnesium salts, such as some of the "high-grade" imported potash salts, are less likely to cause sand drown than muriate or sulphate of exceptional purity.

Prevention.—Sand drown can be prevented by the use of fertilizers in such a way as to prevent magnesium deficiency. The following are the most important features: (1) Very pure forms of potash fertilizers should not be used unless supplemented with other materials containing magnesia. (2) In soils which predispose to sand drown, some fertilizer containing magnesium should be used. (3) When sulphate of potash or sulphate of ammonia is used as fertilizer, the magnesium deficiency may be remedied by the use of magnesium salts or by liming.

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NITROGEN SHORTAGE IN GENERAL

Some plants when deprived of nitrogen are able to grow to maturity and produce blossoms and fruit solely by utilizing the nitrogen that was already stored in the seed, but the aerial growth is very much restricted or dwarfed, in much the same way as in water shortage, or drought injury. In plants dwarfed by drought, the relative size of shoot and root system is approximately normal, while in the case of nitrogen deficiency the root is abnormally elongated. It has been stated that this elongation of the root to many times that of the shoot is an infallible indicator of nitrogen shortage. It has been shown by cultures to be true for corn, and many cases of similar relations have been observed in plants growing in nitrogen-deficient soils under natural conditions. In pronounced nitrogen deficiency, the foliage may assume a light-green to yellowish-green color and with chronic or continued shortage become dry and yellowish brown.

In many cases, nitrogen shortage is expressed only by dwarfed plants and lessened production of the commercial product. The shortage of available nitrogen may cause modified flower development and consequent unfruitfulness. It is one of the factors that influences sterility and is also a factor in the premature dropping of fruit. It is also the belief that biennial or irregular bearing in fruit trees is largely a nutritional problem in which a disturbed nitrogen relation is the most important factor, although certain varieties are more prone to the habit than others.

Quality or other features of the products which do mature may suffer from nitrogen shortage.

YELLOW BERRY OF WHEAT

An undesirable condition of matured wheat grains in which normally hard or flinty grains are partially or entirely starchy in composition has been designated as "yellow belly" or "yellow berry." This condition is familiar to farmers, grain dealers and millers in many sections of the country.

History.—The appearance of wheat grains showing varying degrees of mealiness has long been recognized, such terms as flinty, mealy and half mealy being used, but it is only in recent years that workers have recognized the trouble under the name of yellow berry. It was briefly reported in 1904 by Bolley. In Nebraska and adjacent territory, the deterioration of hard winter wheat caused by the appearance of yellow kernels was of sufficient concern in 1902 to form the subject of a special investigation, the results of which were published in 1905 (Lyon and Keyser). It has been recognized as a factor in the quality of Minnesota wheats, although no specific name was applied to the condition (Snyder, 1904, 1905). More detailed studies of the trouble were published by Roberts and Freeman on the yellow-berry problem in Kansas hard winter wheats (1908) and by Headden (1915) on its cause and prevention in Colorado. Later investigations by Roberts (1919) have contributed additional data on the physiological processes resulting in yellow berry. The trouble has also been given consideration in various bulletins dealing with wheat culture and with chemical, milling and baking qualities of wheat both in the hard-wheat territory of the Plains states and in Pacific Coast sections.

Symptoms and Effects.—Yellow berry cannot be detected by any abnormal condition of the growing crop but is evident only in the threshed grain. The trouble is characterized by

. . . the appearance (in hard flinty wheats) of grains of light-yellow color opaque, soft and starchy. These opaque grains, constituting what are called the "yellow berries," may have this character throughout; but sometimes from a small fraction to a half of a grain will be yellow and starchy, while the remainder of the kernel will be hard, flinty and translucent. The difference in color between the flinty grains and the "yellow berries" is due to differences in the structure and contents of the cells of the endosperm (Roberts and Freeman, 1908).

It very often happens that the only imperfection in a kernel will be a sharply defined spot in one or the other half or in both halves of the kernel; again, the affection is more diffused and may involve one-half of the kernel or a streak along the back of the kernel. Owing to the fact that these spots and areas are less translucent than the surrounding flinty portion, often being quite opaque, the best manner of observing the kernels is by transmitted light. In this way, it will be discovered that many kernels which by reflected light one would consider free from affection are in reality quite badly affected. When the berry is wholly affected, its general color will be affected by the color of the bran or outer coating and will vary from dull white with a tinge of yellow to yellow. Such kernels are usually, if not always, plump and when cut transversely exhibit a white,

starchy interior without any horny portion whatever. Such kernels are soft and starchy. If such kernels as show small yellow spots be cut through transversely, these spots will show in the section as white, mealy or starchy circles embedded in a horny, translucent matrix (Headden, 1915).

The amount of yellow berry has been noted to vary for different seasons in the same locality. During 4 years, the percentage ranged from 4.3 to 25 in Nebraska; none to 42 per cent was recorded for different varieties in Colorado in 1913; while as high as 80 per cent has been reported for Turkey in Oregon.

Grains affected with yellow berry show three pronounced deviations from the normal: (1) a modification of the structure and contents of the endosperm; (2) weight and specific gravity below normal glassy grains of the same variety; and (3) a reduced protein content.

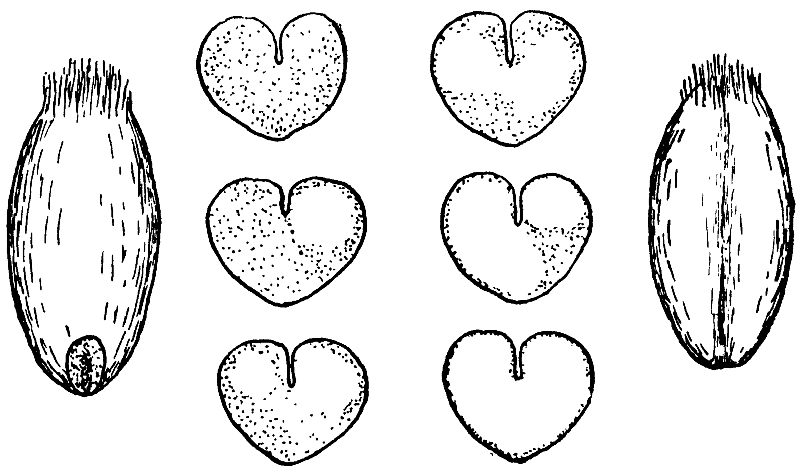

FIG. 31.—Diagrams showing the varying amounts of starchy and flinty endosperm in "yellow berry" of wheat.

The vacuoles are larger and more numerous in yellow-berry endosperm than in normal flinty endosperm and the starch granules are larger, according to Lyon and Keyser (1905), while Roberts (1919) claims that the starch grains of flinty kernels are larger. Analyses have shown that yellow-berry kernels contain a higher percentage of starch, varying from 1 to 4 per cent. The normal berries have a much greater strength than yellow berries, as indicated by crushing tests. The difference in weight is not great, and conflicting results have been obtained: 2.596 grams for 100 yellow-berry kernels and 2.74 grams for 100 hard kernels, an average of 0.0291 gram for yellow berry and 0.0287 gram for hard (Roberts, 1919).

The results of 30 analyses made of flinty and starchy kernels selected from the same sample of wheat show a difference of approximately 2 per cent of crude protein in favor of the flinty berries (Headden, 1915).

Yellow berry is an important trouble, since its presence lowers the grade or quality of wheat due to the generally accepted belief that the presence of the starchy grains lowers the quality of flour that can be produced. For this reason, wheat showing yellow berry sells at a lower price than the hard, flinty wheats higher in gluten. Millers in some sections of the country have referred to wheat affected with yellow berry as deteriorated wheat, thus expressing a detrimental effect. It has been claimed that yellow berry reduces not only quality but quantity of flour because of greater mechanical difficulties in freeing the bran from the floury portions of the starchy grains.

Etiology.—Since the first reports of this trouble, various explanations have been offered for its occurrence. The principal causes which have been suggested are: (1) climatic factors operating upon the grain while in the chaff, either during the last part of the ripening period or after cutting; (2) hereditary tendencies operating independent of environment; (3) disturbed nutrition due to unfavorable water or soil relations. Bolley thought the trouble was due to the action of air, moisture and sun upon the grain and that consequently it could be largely prevented by proper care in harvesting and curing. Lyon and Keyser (1905) attached considerable importance to seasonal variations affecting the time of ripening and concluded that yellow berry increased in amount as the ripeness of the grain increased and also as the length of time of the exposure of the cut grain to the weather was increased, but they also state that "a soil rich in nitrogen and a hot, dry, growing season are, other things being equal, less likely to produce yellow berries even under unfavorable conditions."

Roberts and Freeman (1908) and Roberts (1919) showed that date of seeding affected the amount of yellow berry by affecting the date of ripening, the highest percentage of starchy grains appearing in the later maturing grain. They also suggested (1908) that

> . . . heredity is a strong factor in determining the occurrence of yellow berry in wheat and that pure varieties could probably be isolated that would produce little or no yellow berry.

Later (1919), Roberts concluded from his field tests that

> The operation of common causes for the production of yellow berry overshadowed any differences that may have been due to hereditary tendencies and precludes a definite statement regarding the relation of hereditary tendencies in hard winter wheats towards the production of yellow berry.

It has been claimed by some that excessive amounts of moisture either from natural precipitation or from irrigation increase the amount of yellow berry, but this has not been substantiated by tests. Headden (1915) found no appreciable effect of increased irrigation up to 3 acre-feet on either physical properties or chemical composition of the wheat. It is

also interesting to note that yellow berry was as prevalent on wheat plots which had received 16 loads of well-rotted manure to the acre as on plots which had received no manure. In numerous tests carried out during several years by Headden (1915), the application of sodic nitrate to the soil either prevented yellow berry entirely or greatly reduced it in amount, while the use of a potash fertilizer produced a marked increase in the percentage of starchy grains. This behavior may be illustrated by the following figures showing percentage of yellow berry in three varieties:

	Nitrogen, per cent	Potassium, per cent	Check, per cent
Defiance—1913	0	30	24
Defiance—1914	0	63	36
Red Fife—1913	0	42	31
Red Fife—1914	24	98	98
Kubanka—1913	0	37	31
Kubanka—1914	23	96	97

The use of a nitrogen fertilizer in the early stages of growth gave the higher yields, while applications at heading time showed more reduction in yellow berry (Davidson and LeClerc, 1923).

From these and other results the conclusion was drawn that the determinative factor in the production of yellow berry in wheat is the ratio between available nitrogen (nitric nitrogen) and the available potassium. If this is the correct explanation, it would seem that the lowering of the nitrogen content through cropping, without using methods to restore the proper nitrate balance, is the most important factor in the production of yellow berry. The effect of available nitrogen on the percentage of yellow berry is shown by the results obtained in dry-farming districts of the Pacific Northwest. In Oregon and Washington, early-plowed summer fallow has given little or no yellow berry, while later-plowed summer fallow has produced wheat with a high percentage of the trouble. In the early-plowed land, conditions are more favorable for the work of the nitrifying bacteria, and consequently the amount of available nitrogen is increased and the percentage of yellow berry is correspondingly decreased.

Prevention.—In the light of present information, it seems that the most reliable method of preventing yellow berry or reducing it in amount is by following a cropping system or adopting cultural practices which will keep up the available supply of nitrogen. Although the application of sodic nitrate in amounts of 40 to 80 pounds per acre is a certain preventive, at least under certain conditions, its use in general wheat farming would probably not be justified. The following practices may be followed with profit not only from the benefit to be derived from the

reduction in yellow berry but also from the effect on general maintenance of fertility and increase in yields: (1) the rotation of crops with the inclusion of a legume preceding wheat whenever possible, the exact rotation to be varied for different regions; (2) the use of summer fallow in regions in which legumes cannot be successfully grown, giving special attention to early plowing and sufficient cultivation to provide the conditions favorable for the activities of the nitrifying organisms of the soil.

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POTASH HUNGER

General Effects.—Some plants require a larger amount of available potash than others in order that they may function normally. This is notably true of those crops which are manufacturing and storing large quantities of carbohydrate reserves. The large quantities of potash fertilizers used in certain sections are evidence of the beneficial effects of increased potash. The general effects of shortage of potash may be noted: (1) a reduced photosynthetic activity and consequently a retarded or dwarfed growth of storage organs, such as fleshy roots or tubers, or in cereal crops the development of vegetative structures at the expense of the grains; (2) in woody plants a suppressed or weak development of terminal shoots which may end in a "dieback," as has been shown for both wild species and cultivated fruits; (3) the appearance of yellowish, brownish or whitish spots in leaves at first near the margin and later more general if the shortage continues and is pronounced; (4) the later blighting

of the foliage and premature death if the shortage is not relieved. It has been shown that the amount of stored carbohydrates, such as sugars and starches, is in direct proportion to the amount of available potash. This will explain the weak shoot development of woody plants suffering from potash hunger, since the cellulose which must be formed in the construction of new organs is also a carbohydrate. While dieback may be the end result of a potash shortage, it should be remembered that various other factors may bring about a similar result. The effect of potash shortage can be demonstrated by growing seedlings in a potash-free substratum. After the reserve carbohydrate has been used up, no more can be constructed and growth ceases; but with the addition of a potassium salt, carbohydrate manufacture is resumed and growth continues. It may be noted that in nitrogen or phosphorus shortage the plant generally completes its vegetative period with the production of all organs, vegetative and reproductive, although it remains dwarfed, while in potash shortage the plant reaches nearer a normal size, but blossom and fruit production are either decreased or inhibited and a premature death is the likely end result.

It has also been shown that the addition of potash fertilizers to soils increases their water-holding capacity beyond that of the same soil without the addition of potash. This being true, a soil short in potash might increase the chances of drought injury. It has been reported that root crops which are suffering from shortage of potash are less resistant to decay during rainy periods and that they wilt more readily during hot weather. On this basis, one of the values of a potash fertilizer may be the increased resistance to parasitic inroads.

It has been shown (Nightingale *et al.*, 1930) that potassium is either directly or indirectly essential for initiating nitrate reduction in the plant, a fact which is the explanation for the frequent accumulation of carbohydrates in cases of potash deficiency. In tomato plants, potash deficiency causes inhibition of cambium activity, limits the development of cork cambium, and causes premature death if fruit is present, due to the killing of the growing points by withdrawal of potash into the fruit.

Potash Hunger of Potatoes.—In some important potato sections, it has been a regular practice to use a potash fertilizer. About 1916, as a result of the cutting off of the German supply of potash, a large amount of no-potash fertilizer was used in Maine, Connecticut and other eastern potato sections (Schreiner, 1917; Clinton, 1919; Morse, 1920). At that time, a "new disease" made its appearance which was at first attributed to a fungous invasion but was later shown to be due to the omission of the potash from the fertilizer. The symptoms and effects of this trouble were: (1) a change of color of the plants during July from a normal healthy green to a peculiar bronze or yellow; (2) the wilting and drooping of the leaflets while the stems stood erect; (3) the drooping and wilting

of plants normally green, the stem not having sufficient strength to stand erect; (4) the appearance of discolored areas on various parts of the stems; (5) the formation of a dry, discolored, spongy area involving the stem at about the surface of the ground; and (6) the premature death which always followed these symptoms. This trouble seemed to be most in evidence on the poorer soils or on those which had been heavily cropped with insufficient attention to the use of fertilizers during the preceding years, while in regions in which potash fertilizers had been very generally used for other crops, such as tobacco, the disease was less in evidence. It was also pointed out that the lack of moisture had an important bearing on this trouble, apparently aggravating its severity. This is in keeping with the general principle that potash shortage increases the severity of drought injury. It was the experience that even small amounts of potash in the fertilizer or the application of relatively small amounts of stable manure in addition to the regular fertilizer devoid of potash were sufficient to prevent this "new disease."

Potash Hunger of Tobacco.—The attempts to grow tobacco in certain soils without the use of a potash fertilizer have also given rise to marked symptoms of potash starvation. The plants are more or less stunted, the leaves puckered or with an uneven surface due to the difference in the rate of growth of the veins and the intercostal areas. The peripheral growth is retarded, and, as a consequence, the margins of the leaves and especially the tips curve downward, giving the condition which the farmer calls "rim bound." The affected leaves are also discolored, a chlorosis beginning at the tips and margins of the leaves and advancing inward and downward; the lower leaves show this discoloration first.

Chlorosis due to potash hunger is promptly followed by the appearance of small dead spots in the affected portions of the leaf. As the malady progresses, large areas of the leaf die, especially along the margins, which frequently become ragged and torn (see Sand Drown, Garner *et al.*, 1923).

This phase of potash hunger is popularly called "rim-fire." In potash hunger the chlorotic portions of the leaves have a dull-yellow color with a bronze or copper overcast much the same as in the potato that is deprived of potash, while in severe cases the green portions of the leaf are a darker shade of green than normal. This trouble is not to be confused with frenching, in which the chlorotic foliage is deformed, the leaves characteristically narrowed, thickened and increased in numbers, so that such names as shoe string, strap leaves, sword leaves or rosette have been suggested (Johnson, 1924). For the distinctions between potash hunger and other chlorotic diseases, see the consideration of sand drown (p. 59). If the potash hunger of tobacco is noted in its early stage, relief may be obtained by the application of a readily available potash salt between the rows of the crop.

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CHAPTER IV

DISEASES DUE TO EXCESSES OF SOLUBLE SALTS IN THE SOIL

In the previous chapter, it was pointed out that our crop plants require ten different chemical elements in order that they may make a normal or thrifty development. Seven of these elements are furnished from soil compounds, and mingled with these essential food materials are various non-essential materials.

Natural and Acquired Excesses.—Either food materials furnishing essential elements or compounds consisting of unessential elements or even containing toxic elements may be present in excessive quantities in the soil. It may be assumed that for each crop there is an optimum concentration of each kind of available food material at which the best growth is maintained and that beyond this optimum, conditions become less and less favorable until, with a given concentration, disturbances result which may be expressed in the appearance of symptoms of disease, which with maximum concentrations may lead to death. If we consider the origin of our soils, it must be at once apparent that many native or residual soils in their virgin state may contain excesses which may inhibit or retard growth or even, in spots, entirely exclude all forms of plant life. In addition to the natural excesses, the composition of land under cultivation may be modified by our agricultural practices, such as irrigation, cultivation or the use of fertilizers, so that natural excesses may be increased or new excesses introduced.

General Effects of Soil Excesses or Overnutrition.—Surpluses of available food materials are very frequently accompanied by an abundance of soil moisture, and the type of growth may be influenced by both the nutrients and the water supply. Certain types of growth are, however, directly traceable to overnourishment, the first effect of which is to be seen in an increased vegetative development, a deeper green than normal, more succulent tissues and a retardation or suppression of reproductive functions. Such overstimulated plants are frequently more sensitive to unfavorable climatic factors and fall an easier prey to some parasitic invaders. General overnutrition leading to an excessive accumulation of plastic substances in the plant in proportion to their utilization may lead to pronounced morphological changes. Among these changes may be mentioned *phyllody*, or the transformation of floral organs into leaf-like structures; *petalody*, or the transformation of calyx bracts into petals; *pistillody*, or the change of stamens into carpels; *abnormal*

proliferation, as illustrated by "rose kings," sprouted pears, doubling of composite blossoms and secondary heads of composits; *fasciation,* or the flattening or banding of cylindrical organs; and *spiralism* (see Symptoms of Disease, p. 51). It should be pointed out that this excessive accumulation of plastic food materials is not always the result of overnourishment but that other factors may influence the utilization of plastic materials and lead to similar responses.

Special Surpluses.—The occurrence of specified surpluses in which some particular food element or compound or toxic elements or compounds lead to injuries may be noted. The essential chemical elements most likely to occur in excess are nitrogen and calcium and less frequently aluminum, magnesium, potassium and phosphorus. Two surpluses of special importance are *alkali,* or the excessive accumulation of soluble salts (see Alkali Injury, p. 89) characteristic of arid or semiarid regions; and *soil acidity,* a rather indefinite soil condition, common in humid regions, that is generally corrected by the addition of lime. Renewed emphasis has recently been given to the fact that "infertility in soils may as well be due to the presence of organic substances of biological origin inimical to proper plant development as to the absence of beneficial elements" (Schreiner, 1923). These toxic compounds are not to be looked upon as excretions from normal roots but appear to be produced under abnormal soil conditions or are formed from the decomposition of crop residues under the influence of soil organisms. Non-essential elements may be either inert or toxic; in the former case they produce injurious results by chemical interaction, in the latter, direct effects upon the living substance. Special mention may be made of the injuries resulting from cropping certain soils high in lime or manganese (see Lime and Manganese Chlorosis, p. 75). Recent experiences have emphasized the extreme toxicity of the element boron to our cultivated plants (see Borax Injury).

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EXCESSES OF NITROGEN

Nitrogen is probably the most active of the essential food elements in contributing to general overnourishment of plants. Under natural conditions, it is rarely present in sufficient quantity to cause injury to our crop plants, but the amount may be increased to the danger point by certain farm-cropping practices or by the addition of excessive quantities

of nitrogen-containing fertilizers. The injurious effects of too much nitrogen are well summarized by Lyon and Buckman (1922):

1. *It may delay maturity* by encouraging vegetative growth. This often endangers the crop to frost or may cause trees to winter badly.

2. *It may weaken the straw and cause lodging in grain.* This is due to an extreme lengthening of the internodes, and as the head fills, the stem is no longer able to support the increased weight.

3. *It may lower quality.* This is especially noticeable in certain grains and fruits, as barley and peaches. The shipping qualities of fruits and vegetables are also impaired.

4. *It may decrease resistance to disease.* This is probably due to a change in the physiological resistance within the plant and also to a thinning of the cell wall, allowing a more ready infection from without.

It should be pointed out that the lodging of grain is not the direct result of high nitrogen content of the soil but rather of the shading of

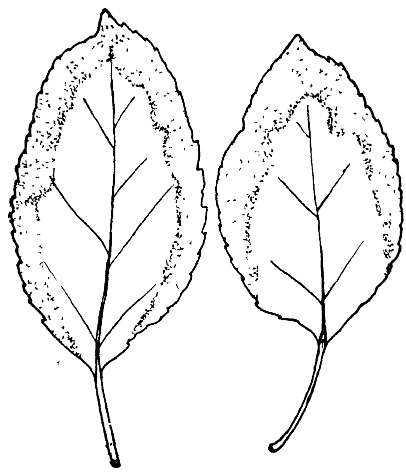

FIG. 32.—Niter burning of apple leaves. (*Drawn after Headden, Colo. Bul.* **155.**)

plants brought about by denser growth, the lack of light being the more important factor in inducing a weak mechanical structure. An amount of nitrogen that may give poor results with a cereal crop may give the highest yields and best quality for certain other crops like many of our garden vegetables.

The desire of gardeners or florists to obtain maximum growth often leads to the excessive use of nitrogenous fertilizers, either compost or commercial products. In case of high concentrations, the symptoms may resemble those of alkali injury, *viz.*, retardation of growth, chlorosis of foliage, followed by burning or browning and a brown, rusty or corroded condition of the root system. The above symptoms appeared in a culture of sweet peas in which the amount of available nitrogen was ten times that of a rich soil of the same type. Excessive use of nitrogen

fertilizers may lead to gummosis and dieback in citrus or stone fruits, or under certain soil conditions the selection of an unsuitable nitrogen fertilizer may be responsible for the injury, rather than the excessive quantity applied. It is stated by Floyd (1917) that

Perhaps most of the cases of dieback which have been classed as acute can be attributed to the effect of ammonia fertilizers added to the soil. Of the different organic sources of ammonia, cottonseed meal and tankage are considered in practice to be more active in bringing on the disease.

Brief mention may be made of the so-called "niter poisoning" of apples in Colorado, which is attributed to a surplus of soluble nitrates in clean, cultivated orchards (Headden, 1910; Leach, 1921) due to pronounced activity of the nitrifying bacteria. In this case, the margins of the leaves first showed a browning or burning.

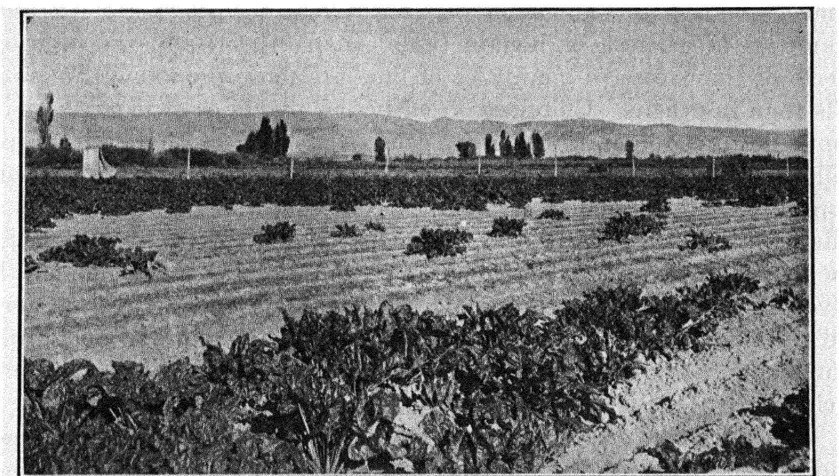

FIG. 33.—A niter spot. (*After Headden, Colo. Bul.* 155.)

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See also GRAEBNER and LYON and BUCKMAN under General References.

LIME OR MANGANESE CHLOROSIS

The failure of plants under normal light conditions to produce the characteristic green pigment or chlorophyll, thus causing yellowing or *chlorosis*, is a symptom of a deranged nutrition. The failure to develop chlorophyll in absence of light is called *eliolation* (see Chap. IX).

The Causes of Chlorosis.—Many different factors may be responsible for chlorosis, which may be an accompanying symptom of either parasitic or non-parasitic troubles. The following more important causes are:

1. Parasitic invasions, either plant or insect.
2. Derangement of nutrition due to lack of essential mineral nutrients, because of either their absence or their non-availability, such as lack of nitrogen, magnesium or iron.
3. Derangement caused by excesses of water or by surpluses of mineral constituents of the soil. The most important chlorotic conditions develop in strongly calcareous or manganese-containing soils or in the alkali soils (see alkali injury) of semiarid country.
4. Low temperatures (see Chap. VIII).

Types of Lime or Manganese Chlorosis.—It has long been known that certain plants, when grown on calcareous soils, develop a sickly chlorotic foliage. Some plants are much more sensitive to the presence of excessive quantities of calcium carbonate (lime) than others, and the injurious effects of the excessive quantities of lime are more pronounced in some soils than in others. Chlorosis of cultivated plants or of native vegetation is not uncommon in the cretaceous soils of the southern Mississippi Valley. Special attention has been given to lime chlorosis of specific crops in certain regions. Grapes, especially American varieties, have suffered from chlorosis in the vineyards of France (Sartorius, 1929). The leaves and shoots are a yellowish green instead of normal green, the leaves remain smaller than normal, the wood does not mature properly and the entire nutrition suffers. Analyses of some soils in which grapes showed the above symptoms gave 18.93 per cent of lime, while soil producing normal vines contained only 1.81 per cent (Graebner, 1921).

The effect of calcareous soils on the culture of pineapples has been studied especially in Porto Rico (Gile, 1911). The plants at different stages or under different conditions showed extreme chlorosis or only moderate effects. Sometimes they were almost ivory white, in other cases yellowish white with streaks of red and green, while in others the new hearts might be creamy white and the outer leaves a light green. Plants showing the extreme type of chlorosis developed brown lesions and finally decayed. It was found that ordinary sandy soils containing as little as 2 per cent of calcium carbonate were unsuitable for pineapples but that "soils composed principally of organic matter may contain about 40 per cent of calcium carbonate and still produce vigorous plants." A similar chlorosis of sugar cane has also been noted in Porto Rico (Gile and Carrero, 1918).

Pineapples have also been seriously affected on the black manganese soils of the Hawaiian Islands. The principal symptoms are a retarded growth, yellowing of the leaves and stunted red or pink fruits, which often crack open and decay. These manganese soils are also toxic to

other cultivated crops, such as corn, pigeon pea, etc., while the guava and various weeds are uninjured. The chlorotic plants are always low in iron, while the unaffected plants contain a normal amount of iron in their ash (Johnson, 1916, 1924).

While the above cases of chlorosis have occurred on soils normally high in lime or manganese, injury to citrus trees has been reported by the application of ground limestone (Floyd, 1917) as a corrective for soil acidity.

The injury to the citrus trees is characterized by a frenching of the foliage, a partial defoliation, the presence of multiple buds on the defoliated terminal branches, a bushy, somewhat rosette-like growth of the terminal branches and a dying back of the branches.

On the same soils, the seedlings of cover crops showed mottling of their foliage, a poor growth and early death. The affected groves were all on dry, sandy lands, lacking in humus. The usual application of lime was 1 to 2 tons per acre, while sometimes a larger amount was used or a number of successive applications were made.

The chlorosis of coniferous seedlings has been a serious problem in a number of nurseries, especially those operated by the Forest Service at Morton, Neb., and Pocatello, Idaho. The yellowing affects the young leaves first, but all the foliage may become chlorotic.

In serious cases, the leaves are short, inclined to curl and are less turgid than normal leaves (as a consequence of lack of sugars and, therefore, low osmotic pressure). The terminal bud either fails to develop or is dwarfed and usually abnormally light in color. The height and diameter of the stem, the length of the roots and especially the ability to form fibrous lateral roots also appear to suffer in typical cases of chlorosis. The disease may occur in patches, or isolated yellow plants may occur. In severe cases death ensues, the parts first discolored being the first to die (Korstian *et al.*, 1921).

It was also noted that the diseased seedlings, although growing less vigorously than normal, continued growth later in the season and were, therefore, more susceptible to winter injury.

Various other fruit or nut plants are sensitive to lime and frequently develop chlorosis under commercial cultivation (Bennett, 1931). Some of these are:

Pear, apple, quince, peach, apricot, prune, plum, cherry, walnut, orange and lemon. Raspberries also suffer severely. Among ornamental plants, eucalyptus, acacia and many other trees and shrubs are known to show chlorosis in high lime soils. Pear trees grafted on Japanese or quince root stocks develop chlorosis on high lime soils much more quickly and severely than do those on French stocks. Stone fruits on myrobalan stocks generally show little or no chlorosis, while peach and apricots on peach stocks may suffer severely.

A type of chlorosis etiologically different from those previously recorded has been studied in Rhode Island.

A chlorotic condition has been noted frequently with various crops—particularly oats, spinach, corn, beets and beans—on soils limed to the neutral point . . . repeated attempts to relate the condition found at this station to an iron deficiency or to correct it by supplying iron in various ways have completely failed (Gilbert *et al.*, 1926).

Etiology.—Lime-induced chlorosis appears to be caused in two different ways. In perhaps the great majority of cases, calcareous or manganiferous soils cause chlorosis by iron starvation. These soils are not deficient in iron, but in lime chlorosis the excess of lime changes the iron compounds into insoluble, colloidal iron which cannot be utilized by the plant or in the manganiferous soils to a much more difficultly available ferric iron. This explanation seems plausible for all those cases in which the normal green color may be restored by spraying the chlorotic plants with iron sulphate. From studies on pineapple chlorosis on manganiferous soils, it was concluded (McGeorge, 1923) that the injury is the result of a greater assimilation of lime due to excess of manganese and to the greater immobility of the iron in the plant due to the excessive lime content of leaves and stems. In other cases in which iron is not shown to be a corrective, analyses have shown the chlorotic parts to contain as much iron as normal green plants and sometimes more (Gilbert, 1926, 1928). The conclusion is drawn in such cases that chlorosis is not due to iron becoming locked up in the soil but rather to a disturbed or inhibited iron metabolism. Treating with small quantities of manganese salts appeared to unlock the metabolic processes and restore the affected plants to their healthy green color.

Prevention or Control.—In cases of chlorosis of the first type, the trouble may be relieved by (1) mechanical application of iron on or in the plant or (2) soil treatment to render the iron available through the root system. Two methods of introducing iron into the aerial parts of plants are available: (1) by spraying the chlorotic plants with a solution of an iron salt and (2) by injection of an iron salt into the wood in trees or other woody plants. Various iron salts such as iron sulphate, chloride or nitrate, ferrous or ferric citrate or tartrate or iron ammonium citrate may be used, but the first is most generally employed. For spraying, solutions of iron sulphate varying from 1 to 5 per cent have been recommended. Spraying with 1 per cent ferrous sulphate has been adopted as a part of the regular nursery practice in several of the forest-service nurseries (Korstian *et al.*, 1921). Spraying for the control of chlorosis of pineapples on the manganiferous soils of Hawaii has proved commercially feasible, about 10 pounds per acre at intervals of 2 to 4 months being effective (Johnson, 1916). Spraying for fruit-tree chlorosis has not proved to be commercially successful (Bennett, 1931). For injecting iron into the wood, two methods are employed: (1) filling one to several bored holes in the wood with dry iron powder, after which the holes are sealed

with wax (Wann, 1930; Bennett, 1931); and (2) the injection of a solution of the desired salt from a reservoir attached by a rubber tube to a threaded pipe inserted into the bore hole, which should extend two-thirds to three-fourths the diameter of the trunk and slightly downward. One hole is sufficient for trees up to 6 inches in diameter, with two or more for larger trees. One ounce per gallon may be used, with dosage varying according to size of tree or dormancy. According to Bennett (1931), ferrous and ferric citrate have given the best results.

Under some conditions, the iron salt may be added to the soil. Best results with this practice may be expected if the soil is not too strongly alkaline. The iron salt may be added by the *trench* method or by dropping it into holes bored into the soil. For details and dosage, reference should be made to the work of Bennett (1931). In strongly alkaline soils, the iron is prevented from going into solution; hence it would seem desirable to improve the soil conditions by modifying the soil reaction to become acid or at least less alkaline. The use of heavy applications of barnyard manure or commercial fertilizers, such as ammonium sulphate or sulphur, may be expected to be beneficial.

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SOIL-ACIDITY MALNUTRITION

The soil reaction is recognized as having an important influence upon the nutrition of plants. Two opposite reactions must be considered: *alkali* or soil alkalinity and *soil acidity*. Alkali injury is confined largely to arid or semiarid regions, where conditions favor the concentration of soluble salts; while soil acidity is common mainly in regions of abundant rainfall. Acidity as applied to the soil indicates a condition that can generally be corrected by the addition of lime. Soils may be acid, neutral or alkaline in the reaction, and it is well known that the acidity of the soil has a marked effect upon the character and distribution of native vegetation (Wherry, 1920) as well as causing malnutrition in some cultivated plants which are subjected to a soil reaction to which they are unsuited. Of our cultivated plants, some require an acid soil for their best development; others are simply acid tolerant, being able to make a very good growth in acid soils; while others are unable to adjust themselves to acid conditions and if planted in such an environment either make a sickly growth or succumb.

The Origin of Acidity of Soils.—The acidity of a soil may be brought about in a number of different ways: (1) by the addition of manure or sewerage, the deposit of combustion products or the absorption of smoke gases (see Chap. X, Smelter Injury); (2) by the continued use of acid mineral fertilizers like acid phosphates or of others such as sulphur or sulphur salts that are oxidized to form acid; (3) by the interaction of the natural residual components of the soil; (4) by the formation of organic or humic acids by the decomposition of plant remains; and (5) by the removal of lime or other neutralizing bases by plant growth or by leaching from heavy rainfall. It has been pointed out that there are soils which although giving an acid reaction contain neither free acid nor humic colloids. This has been explained by a classification of the types of acidity (Schuckenberg, 1924).

Kinds of Soil Acidity.—The four following types of acidity have been recognized: (1) active acidity; (2) selective absorption by humic compounds; (3) exchange acidity; and (4) hydrolytic acidity. Active acidity is characteristic of uncultivated moor soils but is rare in mineral soils and is due mostly to H_2SO_4. The second is characteristic only of humus soils and shows the formation of free acid by treatment with a neutral salt solution. Exchange acidity is common on all soils poor in lime, and an extract with a neutral salt solution shows an acid reaction due to the

exchange of the trivalent ions of aluminum or iron for the cation of the neutral salt. A water extract, however, shows no acid on titration. Hydrolytic acidity may occur in any soils showing one of the other types of acidity or alone in certain soils poor in lime and rich in humus. In this type, the soil has the capacity to absorb a part of the bases as a result of hydrolysis of salts and thereby set free an equivalent quantity of acid. Hydrolyzable salts are those with a strong base and a weak acid radical and show dissociation in aqueous solutions.

Injurious Effects of Soil Acidity.—While the exact response of acid-sensitive plants will vary with the species and with the kind of soil acidity, the general effects are quite similar. The first effect with moderate acidity will be *retarded growth* and a *pallor* or less intense green than normal, which if the unfavorable conditions continue may become more pronounced. Foliage may become *mottled,* showing lighter-green areas between the veins, or the *chlorosis* may become diffuse or general. Such affected plants may weaken and die prematurely, or growth may be resumed if rains modify the acidity later in the season. Roots of affected plants make a poor development, and many of the lateral feeders may be repeatedly killed back. It is a significant fact that certain soil organisms are distinctly favored by acid conditions—*e.g.*, the pathogene of club root of cabbage.

Etiology.—The injurious effects of acid soils may be accounted for in a number of different ways: (1) by unfavorable H-ion concentrations; (2) by the direct effects of toxic elements, such as aluminum or magnesium, which appear to occur in the active form when the soil reaction or H-ion concentration is beyond neutrality; or (3) by the non-availability of certain essential nutrients or their reduction in amount, or the elimination or suppression of the supporting or auxiliary action of nutrients, so that normal absorption does not take place. It has been shown that both field and truck crops may be greatly injured by growing under soil conditions which have produced high acidity. In some cases, the unfavorable condition has been produced by the yearly use of fertilizers which have gradually increased the soil acidity until it has become a limiting factor in the growth of truck crops in the same soils (Harter, 1909). Recently, injury to cotton has been reported (Taubenhaus and Ezekiel, 1931) as a result of extreme soil acidity following excessive applications of sulphur. Acidity injury to cereal crops under field conditions has been reported by Ludwigs (1923), and wheat, oats, barley and rye have been tested in cultures (Schuckenberg, 1924).

It is known that the H ion is very toxic to the meristematic tissue of root tips. Recent researches have left little doubt that much of the injury from acid soils may be due to aluminum which is brought into soluble form (see Aluminum Toxicity). Excess iron salts or magnesium may be playing a part also. The shortage of calcium in an acid soil may

operate in two ways: (1) by depriving the plant of the required amount of this necessary element or (2) by preventing the absorption and utilization of other necessary elements such as potassium. Acid soils are also frequently short on nitrogen or even phosphorus. The retarding effect of soil acidity upon the processes of nitrification should also be taken into consideration. It must be evident that soil acidity is a rather complex phenomenon and that the injuries which result are not due to any single factor.

Control.—The acidity of a soil may be corrected by the addition of compounds which will furnish the necessary bases to combine with the acids. Calcium is generally selected because it is cheap and effective. Potassium is too expensive, and magnesium may sometimes be harmful. Agricultural lime in quantities greater than 1 ton per acre is seldom economical but up to 2 tons may be used. It is perhaps best to use the minimum amount required for a given soil condition and employ other practices to build up and maintain general fertility.

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BORON INJURY

Investigations during the last few years have established the fact that boron in very small quantities is indispensable to the growth of

some plants (Brenchley and Warington, 1927) but that this element is extremely toxic if its concentration is but slightly increased.

Historical Statement.—This element when present in small quantities has been reported to exert a stimulating effect on plant growth, acting as a catalyzer rather than as a plastic ash constituent (Agulhon, 1910), but certain injurious effects were noted when larger quantities were available for absorption (Cook, 1916; Cook and Wilson, 1917, 1918). This more recent impetus to the study of the effect of boron on plant growth was due to the tests of borax (sodium salt of tetraboric acid) and calcined colemanite (calcium salt of boric acid) as larvacides for the killing of house flies in horse manure. The studies were made to determine the effect of such treated manure on plant growth, and it was shown to cause injury if used in large amounts.

Boron is widely distributed in soils but not generally in sufficient amount to cause injury. It has been shown that borax may be introduced with impure commercial fertilizers in sufficient amount to cause serious crop injury. The first report of this injurious effect of borax in fertilizers was by Conner (1918), who reported serious injury to corn by a fertilizer which contained 1.92 per cent of this salt. It was soon found that the source of the trouble was the potash salts from a single American locality, Searles Lake, California, which had been put upon the market when the war excluded the standard German salts. The injury from this Searles Lake potash when used on various potash-deficient soils in a number of states led to investigations by other state and Federal workers (Morse, 1920; Plummer and Wolf, 1920; Schreiner *et al.*, 1920). Special attention was given to the effect on potatoes, tobacco, cotton and corn under field conditions and in greenhouse cultures. Later studies were concerned with the above crops and also with beans (Neller and Morse, 1921; Skinner *et al.*, 1923).

More recently, Kelley and Brown (1928) and Schofield and Wilcox (1931) have shown that boron occurs in certain soils and irrigation waters of southern California in sufficient amounts to cause severe injury to trees, while Haas (1929) working under controlled conditions has produced symptoms in the leaves of citrus similar to those found in certain California orchards.

Symptoms and Effects.—The behavior of plants subjected to the action of boron varies with the concentration of this salt and with the soil and moisture conditions. The principal effects are as follows: (1) retardation or prevention of germination; (2) death of plants or stunting so as to give imperfect and uneven stands, with much variation in the size of plants of the same age, both tops and roots being affected; (3) absence of normal color marked by the bleaching or yellowing of normally green parts, especially the margin and tips of leaves, followed by tip burn in the more severe types of injury; (4) reduced growth and premature ripening, with lessened yields. The injury may vary from slight disturbances which are largely outgrown to complete failure of a crop when large amounts of borax are applied.

On Potatoes.—The first evidence of injury in the field is "missing hills" and stunted plants, with but few that approach the normal in size and color. The foliage characters of affected plants were as follows:

There was considerable yellowing of the leaves, more particularly of the margins. This was the most prominent on the more dwarfed and more severely

injured plants. The yellowing was of a bright golden color and not the pale, sickly yellowing usually present in plants that are normally or prematurely ripening (Morse, 1920).

In milder cases, the yellow is confined to the very narrow bands at the leaf margins, and this may be the only indication of injury in plants that are not stunted. Under certain conditions, these symptoms may

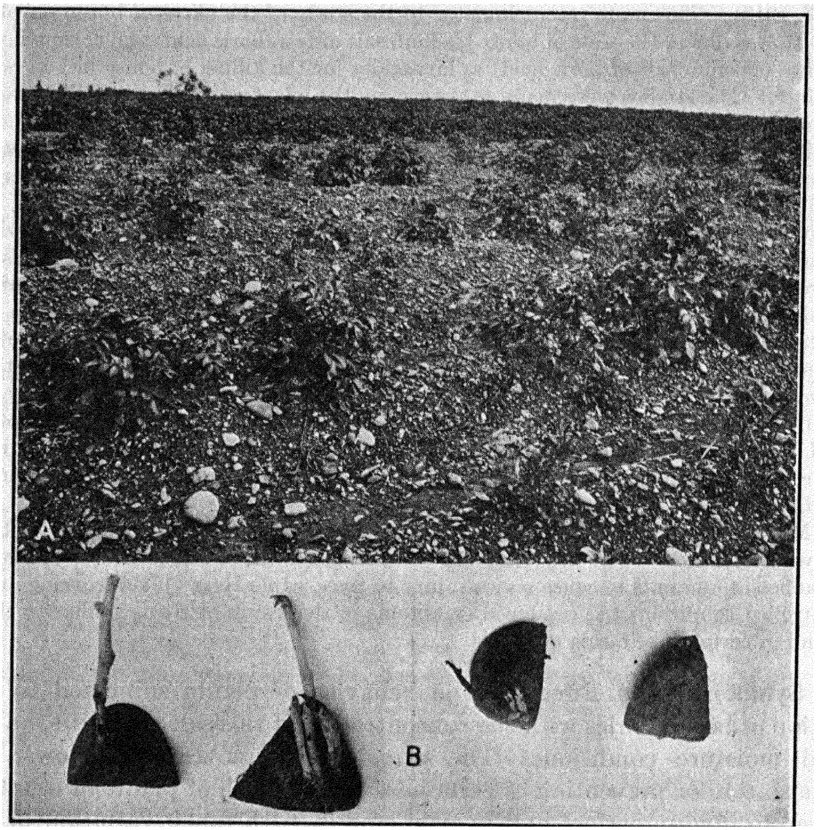

FIG. 34.—Borax injury of potatoes. *A*, potato field showing injury due to use of a potash fertilizer carrying 33 pounds of borax per ton. A no-potash fertilizer used on field in the background; *B*, potato seed pieces from apparently missing hills, showing various stages of borax injury. (*Taken from U. S. Dept. Agr. Circ.* **84**.)

disappear or be masked by the growth of the plants. The injury to parts below ground was variable and consisted of brown stem lesions, complete cutting off of stems, browning and killing of roots, and burning and corrosion of the cut surfaces of the seed pieces, the effects much resembling Rhizoctonia attacks.

According to Morse (1920):

Numerous cases have been reported where yields were not over half or one-third of a normal crop, and some of the more severely injured fields would hardly produce a sufficient crop to pay the cost of harvesting.

On Corn.—The presence of borax may cause delay in germination and distorted and bleached plants, or in severe cases the stalk may cease growing and wither after the young plant has pushed through the soil. In still more serious cases, the seedling lacks the vitality to push above the soil and withers and dies. The injury is during germination and early growth, and the bleaching will vary with the conditions and the amount of borax present in the soil. The amount of discoloration will vary from slight to almost complete bleaching, and affected leaves may be uniformly discolored or they may be banded or streaked with white or yellow. Discoloration may be followed by tip burn or complete wilting without recovery, or, under more favorable conditions, plants showing the trouble when young may recover and make a growth that is nearly normal. The amount of actual injury has varied. When heavy rains followed the application of the borax (5 to 10 pounds per acre), there was an increase of both stover and ears, while the same quantities under drier conditions caused very pronounced decreases in yield.

On Cotton.—Borax may cause the death of young seedlings, or they may be stunted and show a weak, slender growth. The death of young plants and the retardation of growth of others may give a poor stand and an uneven growth.

> The seedlings which have not made sufficient growth to put out the first pair of true leaves may remain with no apparent growth for 2 or 3 weeks; then dead areas may appear between the veins and along the margins of the cotyledons or seed leaves. These cotyledons at length become dry, and the plant perishes (Plummer and Wolf, 1920).

Plants less severely injured may show yellowing of the foliage, a stunted growth, cup-shaped leaves, an early shedding of the lower leaves, earlier maturity of the entire plant and very much reduced yields. Affected plants develop fewer and shorter roots than normal plants.

On Tobacco.—Heavy applications of borax (15 pounds or more per acre) to plant beds either inhibit germination or produce yellowish-white, dwarfed plants. Transplants to fields fertilized with borax-containing fertilizers may die within a few days, and those

> . . . which survive either fail to "grow off" or remain stunted in both top and root. When affected plants start to grow, the lower leaves are paler green than normal and are also thicker and less broad. The tissues most distant from the principal veins are palest and may become dead and dry. The leaf margins and tips are rolled downward and at length become "rim bound." The roots of plants which are severely stunted tend to be densely clustered near the end of the main root and are all short and fibrous (Plummer and Wolf, 1920).

Those less injured may have a second group of roots near the surface of the soil, with few intermediate roots. "Firing" of the leaves rather than

normal ripening is characteristic of affected plants as they approach maturity. Both quantity and quality of the product are affected.

A

B

FIG. 35.—Borax injury of corn. *A*, plat to which no borax was applied; *B*, plat receiving 30 pounds of borax per acre. (*After U. S. Dept. Agr. Bul.* **1126.**)

On Beans.—This crop is especially sensitive to borax, even 4 pounds to the acre causing severe injury. The most severely injured seedlings failed to break through the soil, while others which did come up were

chlorotic from the start and the cotyledons did not emerge from the seed coats. In plants which did become established,

. . . the injuries due to borax appeared first on the margins of the first two leaves, particularly the tips. In the larger borax applications, the entire leaf soon turned yellow, then white, followed by a killing of the tissues which progressed from the margins inward. New leaves either failed to appear or were much stunted and almost devoid of chlorophyll (Neller and Morse, 1921).

When seedlings came in contact with the fertilizer, the tap root was killed, and in older plants the root tips of laterals were also killed. With increasing amount of borax, there was a corresponding decrease in the root systems and more browned and killed laterals. Even with only 5 pounds of borax per acre, reductions in dry weight to the extent of 46 per cent were suffered, while complete failure resulted with the higher applications (20 pounds).

On Citrus.—The effect of toxic quantities of boron on citrus includes yellowing or chlorosis of the leaves, sometimes with a very pronounced mottling. Burning or necrosis at either tips or margins is common if the toxic action continues. Dark lesions may also appear removed from the margin. These changes may be accompanied with crinkling and rolling of the leaves, and premature abscission may lead to defoliation. When nitrate of soda was supplied with the toxic quantities of boron, the effect on Valencia oranges was not very different from true mottle (Haas, 1929). Similar injuries were produced in cultures of walnut, pecan, avocado and peach, with considerable necrosis between veins in the walnut. Lemon seedlings were more sensitive than orange seedlings.

Etiological Considerations.—It has been shown by both field and pot cultures that boron in small amounts either alone or when mixed with fertilizers will cause injury to many crop plants, the amount of injury depending on the manner of application, the crop concerned, the amount of the poison applied and the actual concentration of this in the soil water. In field crops, injury has been greatest when the borax or the borax-containing fertilizer was applied in the drill row, rather than broadcast. This variation may be illustrated by beans, with 3 pounds per acre as the toxic limit when put in the drills or 5 pounds per acre when broadcast. It has also been shown that there is less injury to potatoes from equivalent amounts of borax when the fertilizer is applied in the furrow some time previous to planting rather than at the time of planting. Borax injury is more pronounced in sandy than in clayey soils.

The presence of iron and aluminum in clay soils and the ability of borax to form insoluble compounds with these elements, especially under certain conditions, may in part account for the differences which appear in these two soil types. It is believed, however, the phenomenon of colloidal absorption is an

important factor in accounting for the tolerance of plants to larger amounts of borax on clay soils (Plummer and Wolf, 1920).

Greenhouse tests and field observations have shown that the injury from borax is affected by the amount of soil moisture. When heavy rains follow immediately after planting, the danger of injury is much lessened due to the leaching action of the soil water. In controlled tests, it was shown that "given amounts of borax which were toxic toward germination and growth in soil held at a 30 per cent moisture content were more toxic in soil containing half as much water" (Neller and Morse, 1921). The leaching of borax by rains has an important action in preventing the residual effect of this compound. It has been shown by planting tests that the toxic effect does not persist to the next season, and the recovery of plants as the season advances may also be due to the leaching out of the borax.

The toxic limit for wheat is 2 to 3 pounds of borax per acre; for beans, 3 pounds; for potatoes, 5 pounds; for cotton, 8 to 9 pounds. These quantities are small compared with the amounts of arsenic or copper which such plants will endure. The poisonous action of borax must be due to the element boron which it contains. A number of theories have been advanced to explain the toxic action of boron: (1) that it is *antizymotic, i.e.*, that it prevents the formation and action of enzymes or ferments which are vital to the process of germination and growth; (2) that it interferes with the translocation of carbohydrate food by forming chemical union with sugars and related compounds; and (3) that it interferes with the formation of chlorophyll by withholding iron from the tissues and so induces chlorosis. Whatever its mode of action, it is certain that minute quantities of boron exert a profound influence upon the nutritive processes and the regeneration of the chlorophyll.

Prevention of Borax Injury.—Since impure fertilizers are the source of borax in sufficient concentration to cause crop injury, protection lies in the use of fertilizers which have been purified or in which the borax content is below the danger point for the amount of fertilizer that is ordinarily used per acre. Past experiences have resulted in commercial concerns giving greater attention to putting pure, high-grade products on the market. The recent investigations in California suggest that some consideration may need to be given to the purity of the irrigation water. Boron toxicity in cultures was somewhat relieved by the addition of various amounts of ferric sulphate. Leaching appears also to overcome the toxicity.

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ALKALI INJURY

When one thinks of alkali in its relation to natural vegetation or to crop production, a picture is presented of barren soil either devoid of plant life or supporting only a sparse and dwarfed plant cover. This picture represents the extreme of the alkali effect, which in many cases may be only slightly in evidence. As a purely chemical concept the word "alkali" refers to substances having a basic reaction, but as applied to soils and the growth of some plants it refers to the natural accumulation of soluble salts in such concentration as to cause injury. The very substances which constitute the alkali salts may stimulate growth when present in the form of dilute solutions. It is then the concentration of these salts, rather than the kind or quality, that is primarily responsible for deleterious effects of alkali soils.

The Composition of Alkali.—Soils that are alkali may include the chlorides, sulphates, carbonates, bicarbonates, phosphates and nitrates of the common bases sodium, calcium, potassium, magnesium and sometimes ammonia. The actual constituents in a given locality are, however, variable, but the three principal ingredients are (1) sodium chloride, or common salt; (2) sulphate of soda, or Glauber's salt; (3) and the carbonate of soda, or sal soda. The chloride and sulphate of sodium and other bases may become concentrated at the surface of the soil and produce a whitish incrustation, characteristic of what is sometimes called "white alkali." These alkali spots are especially noticeable in semiarid lands and become most conspicuous during the dry periods. The carbonates of the bases, but especially the carbonate of soda, are capable of dissolving the organic matter of the soil, and the solution and the surface accumulations are

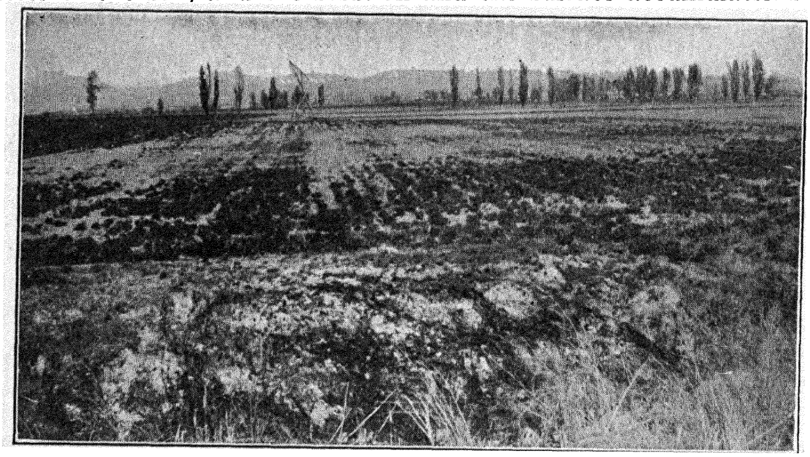

FIG. 36.—Alfalfa being killed by alkali brought to the surface by a rising water table. (*Photograph by F. J. Sievers.*)

dark, hence the popular name of "black alkali." Both white and black alkali are injurious to vegetation, but the latter is much more destructive, as would be expected from the nature of its action on soil humus.

Symptoms and Effects of Alkali.—The effects of alkali will vary according to the concentration and kind of the salts present and the resistance or tolerance of the plant to alkali salts. It seems to hold true that most of our valuable crop plants are rather sensitive to alkali and so refuse to grow in soils that sometimes support a conspicuous cover of native alkali-resistant plants.

The first effect of alkali that may be noted is retardation or prevention of germination of seeds. In strong alkali soils, seeds may remain dormant, since the physiological processes of germination cannot be initiated. Such seed will frequently grow when removed from the alkali soil to conditions of moisture and temperature that favor germination. In soils with much alkali, seedlings may succumb after reaching a few inches

in height. In others with less alkali, germination may be very greatly retarded and the young plants make a sickly, slender growth, marked by chlorosis and early death, without reaching flower and fruit production. In more resistant plants or in less concentrated alkali, the growth may be retarded and chlorosis may be manifest, but the crop may reach fruiting maturity, sometimes with burning of the foliage as the season advances. The general effects upon the plant and upon its producing power are quite comparable to drought injury. It should be understood that many other environmental factors besides alkali may cause chlorosis of the foliage, such as excess of lime, shortage of magnesium, low temperatures, etc., but

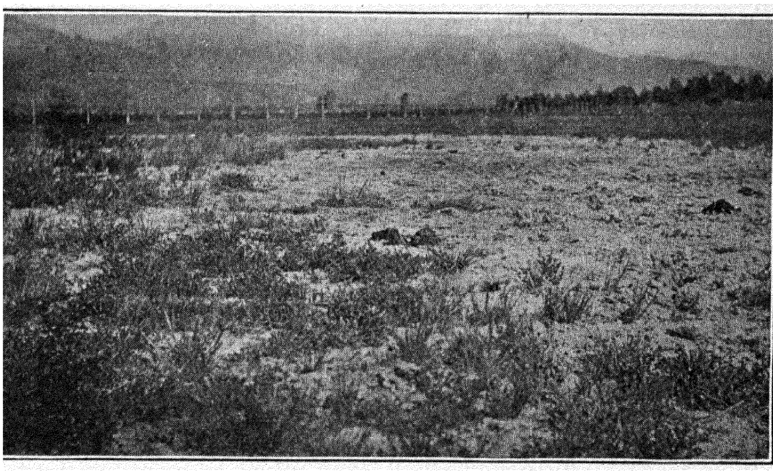

FIG. 37.—An alkali spot. Note the absence of a plant cover. (*After Harris.*)

a careful study of the soil conditions and surrounding vegetation will generally yield a reliable diagnosis. It may also be noted that some plants may suffer from alkali without the warning symptom of chlorosis, the first marked evidence being a wilting of the foliage. The above considerations apply especially to annuals or herbaceous perennials.

Shade or orchard trees may be planted in soils with a relatively high content of alkali salts and make a good growth for a time until the alkali becomes more concentrated at the surface. The effect on established trees is a retarded growth and more or less chlorosis. In some cases, the foliage may become a brilliant golden yellow. In fruit trees, such as apple, apricot, etc., the trees may be dwarfed, the foliage scanty and the new shoots short, with few small leaves. Such trees may survive for a period of years in this weakened condition, or, with increase in the concentration of the alkali, they may succumb. The burning or blighting of leaves at the tip or margin is not an uncommon symptom of alkali injury, and these symptoms may be followed by premature fall. The gradual accumulation of the salts at the surface of the soil may cause a

corrosion of the bark at the crown, resulting, in extreme cases, in a girdling which interrupts the downward movement of elaborated food to the root system. The general effects will then be very similar to the crown or collar rot due to winter injury, and one set of conditions may supplement or aggravate the other. This corrosive action of alkali at the surface of the ground is most evident in the case of black alkali but generally of minor importance in contrast with the general interference with the nutrition.

Plants that have become adjusted to saline conditions have undergone pronounced structural modifications, and it has been shown that these changes of structure are very similar to those adopted by xerophytic or drought-resisting vegetation; *i.e.*, they are designed to diminish the evaporation or transpiration. Some of these xerophytic modifications are

> . . . diminution in size of leaves, assumption of cylindrical or spinous forms, sinking in of the breathing pores below the outer surface, dense hairy covering, resinous exudations, etc. Internally we find that xerophile plants have developed on their upper or outer leaf surfaces, instead of one, several layers of palisade cells (Hilgard, 1906).

When our crop plants are forced to endure alkali, some modifications of structure result. Harter (1908) found that wheat, oats and barley grown in saline solutions developed a conspicuous bloom or waxy covering on the leaves, a thicker cuticle and external epidermal walls and smaller epidermal cells. If our crop plants could modify their structure more rapidly under the influence of alkali, they might be more resistant.

Etiological Considerations.—Alkali causes injury to our crop plants in a number of ways: (1) by the reduction, or the prevention, of absorption; (2) by a reduction of transpiration; (3) by toxic action upon the living cells; (4) by interference with the chlorophyll apparatus and the process of photosynthesis; (5) by corrosive action upon roots or stems in contact with the concentrated solutions, especially sodium carbonate; and indirectly (6) by affecting the physical properties of soils and their biological activities.

Seeds fail to germinate in strong alkali, because they do not imbibe or absorb the necessary moisture.

> It may be said, roughly speaking, that the absorption of water by the roots begins to diminish so soon as the concentration of the saline solution approaches or exceeds one-half of 1 per cent; while when it rises as high as 3 per cent, water absorption ceases by the roots even in the wettest soils, and the plants suffer from drought quite as much as from any directly injurious effects of the salts (Hilgard, 1906).

The total amount of the salts found in alkali lands varies from 0.1 to 3 per cent of the weight of the soil taken to a depth of 4 feet, so we may

note that the concentration of the soil water may be such as to retard or prevent absorption. If the cell sap of the root cells is more concentrated or has a higher osmotic value than the soil water, there will be a movement of water into the cells; but when the concentration within and without the cells begins to be equalized, absorption is gradually retarded, until, with a greater concentration of osmotically active solutes outside the cell, water is actually withdrawn and the protoplasm contracts from the cell wall. This plasmolysis of the cell would be the final effect of strong alkali, and such plasmolyzed cells die unless the osmotic balance is soon restored. It is not solely water absorption that is interfered with, but the alkali salts, especially sodium, interfere with the absorption and utilization of necessary nutrients.

It has been shown by certain tests that minute quantities of alkali salts stimulate transpiration, but when they are present in sufficient quantities to bring about modifications of structure, transpiration is much retarded, and consequently growth is less.

According to Harris (1915):

> The anion, or acid radical, and not the cation, or basic radical, determines the toxicity of alkali salts in the soil. Of the acid radicals used, chloride was decidedly the most toxic, while sodium was the most toxic base.

The opposite condition, *viz.*, the greater influence of the cation, was found for solution cultures by Kearney and Cameron (1902). Many experiments have shown the toxic action of single salts when present in solution cultures and the reduction of this toxicity by the presence of other elements. These same salts are not of necessity toxic when added in the same quantity to soil. The results obtained by Harris (1915) were based on soil cultures to which the different salts were added. He gives the following order of toxicity for soluble salts in the soil:

> . . . sodium chloride, calcium chloride, potassium chloride, sodium nitrate, magnesium chloride, potassium nitrate, magnesium nitrate, sodium carbonate, potassium carbonate, sodium sulphate, potassium sulphate and magnesium sulphate. The injurious action of the alkali salts is not in all cases proportional to the osmotic pressure of the salts.

Judging from the frequency with which chlorosis is the result of alkali, the retardation or slowing up of the photosynthetic activities should receive more emphasis as a phase of alkali injury. This alkali chlorosis is apparently connected with iron and calcium nutrition, since the solution of both is prevented by black alkali (Hibbard, 1925).

Alkali salts affect crop growth indirectly by modifying the physical properties of the soil, causing the "freezing up." The first effect to be noted is the puddling of the soil or deflocculating of the particles, producing a compact condition which prevents the rapid rise of water, while

the more active condition is seen in the firm, hard crusts which form on the surface of the soil, thus affording mechanical interference with plant growth.

Under the surface of many soils in the arid regions, particularly in sections of abundant alkali, a hard layer is found which obstructs the penetration of both roots and water. Hardpans are not always caused by alkali but are more likely to be formed if it is present (Harris, 1920).

The physical effect of alkali on soils in leading to unproductiveness is stressed (Breazeale, 1927) as being fully as important as the toxic properties of the salt solutions, as illustrated by barren alkali soils which are non-toxic.

Alkali soils have a marked effect upon the life and activities of soil organisms, especially the nitrifying and nitrogen-fixing bacteria, decreasing their activities. Just how much importance should be attached to the modification of the biological activities of the soil by the presence of alkali is still somewhat uncertain.

Resistance to Alkali.—There are great variations in the tolerance of plants to alkali, some being able to grow in excessive alkali (above 1.5 per cent), while others are barely able to grow in weak alkali (0 to 0.4 per cent). This variability is well illustrated by the following list showing the plants that are likely to succeed in various concentrations:

Excessive alkali (above 1.5 per cent)—Salt bushes and salt grasses.

Very strong alkali (1.0 to 1.5 per cent)—Date palm and pomegranate bushes.

Strong alkali (0.8 to 1 per cent)—Sugar beets, western wheat grass, awnless brome grass and tall meadow oat grass.

Medium-strong alkali (0.6 to 0.8 per cent)—Meadow fescue, Italian rye grass, slender wheat grass, foxtail millet, rape, kale, sorghum and barley for hay.

Medium alkali (0.4 to 0.6 per cent)—Red top, timothy, orchard grass, cotton, asparagus, wheat and oats for hay, rye and barley.

Weak alkali (0.0 to 0.4 per cent)—Wheat, emmer and oats for grain, kafir, milo, proso millet, alfalfa, field peas, vetches, horse beans and sweet clover (Kearney, 1911).

The relative resistance of some of the common crops in the seedling stage is given by Harris (1915) in the following order: barley, oats, wheat, alfalfa, sugar beets, corn and Canada field peas.

Fruit crops also show great variation in their tolerance to alkali, and some varieties of a given fruit may show pronounced differences in resistance to alkali injury (Kelley and Thomas, 1920). Grapes are listed as the most tolerant to alkali, being able to withstand soils containing 45,760 pounds of total alkali per acre (surface 4 feet), while the highest amount endured by the mulberry without injury was 5740 pounds per acre. The comparative resistance to total alkali consisting of Glauber's salt, sal soda and common salt has been given in the following order: grapes, olives, almonds, figs, oranges, pears, apples, prunes, peaches, apricots, lemons and mulberry (Loughridge, 1901).

Prevention of Alkali Injury.—Alkali accumulation is characteristic of semiarid lands in which the high evaporation brings the alkali salts to the surface, while there is not sufficient precipitation to redistribute them in the soil. Excess of soluble salts is sometimes sufficient in long-continued ground culture under glass to cause crop injury (Connor and Gregory, 1928). Irrigation favors the surface accumulation of alkali in semiarid regions, since alkali previously well distributed in the soil may be brought to the surface with the rise of capillary water and left as a surface incrustation when the moisture evaporates. This is what is known as the "rise of alkali." The purity of the irrigation water, *i.e.*, its content of alkali salts, is of importance, as the alkali accumulation will be accelerated by the rise of impure water.

No single practice will handle the difficulties encountered in cropping alkali lands. It may be noted first that lands favoring alkali injury need heavier irrigation than lands in which there is no alkali problem. Also that irrigation water carrying much alkali must be used more copiously than purer water. The principal methods of handling alkali lands so as to prevent crop injury or to reduce it to a minimum are as follows:

1. The use of alkali-resistant or alkali-tolerant crops.
2. The adoption of cultural practices that will keep the alkali well distributed in the soil or retard or delay its accumulation at the surface. The most important practices are those which retard evaporation, of which the following may be mentioned: (*a*) cultivation to keep a surface soil mulch; (*b*) the use of a surface mulch of manure, straw, leaves or sand; (*c*) the establishment of a crop cover that will shade the soil.
3. The burying of the surface soil by deep plowing.
4. The lowering of the water table and the prevention of seepage by the use of cement-lined ditches.

The above are, in the main, methods designed for lessening the injury in lands that are moderately alkali. When the alkali accumulation is excessive, the actual removal of the salts from the soil or their transformation into less injurious forms is of most value. Several different practices have been recommended, some of which are of only minor importance. Removal of the surface alkali by scraping or brushing from the surface is rarely practical. Removal by cropping to a heavy alkali feeder like sugar beets has been recommended for lands showing medium-strong alkali to fit them for a more sensitive crop. The most important practices for alkali removal or transformation are: (1) underdrainage alone or supplemented by flooding and leaching; (2) diking and flooding to a depth of several inches, followed by surface drainage of the water and dissolved salts; (3) the neutralization of the sodium carbonate or "black alkali" by the addition of gypsum, which results in the formation of calcium carbonate and sodium sulphate, thus changing the alkali to a much less harmful salt. The use of the gypsum will also facilitate drain-

age and reduce the loss of organic matter, when flooding or underdrainage is practiced (Catlin and Vinson, 1925; Burgess, 1928). Other chemical treatments have also been suggested, such as the application of elemental sulphur (Hibbard, 1921) or small amounts of sulphuric acid. Following the completion of leaching, heavy applications of manure are necessary to restore the land to productiveness. The danger of removing plant-food materials as well as the alkali salts or the creation of detrimental ratios of essential elements as a result of flooding or leaching, and thus rendering lands unproductive for a period, has been given emphasis (Greaves *et al.*, 1923; Hibbard, 1925).

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IMPORTANT DISEASES DUE TO EXCESSES OF SOLUBLE SALTS

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Soil-acidity malnutrition.—(See special treatment, pp. 80–82.)

CHAPTER V

DISEASES DUE TO UNFAVORABLE WATER RELATIONS

Before considering the ways in which a disturbed water relation may affect the growth and production of crop plants, a brief outline of the function of water in the life of plants may be presented.

The Function of Water.—The uses of water are as follows: (1) It serves as the *solvent for, and vehicle of, transport of food and food materials* from the soil into the plant and from cell to cell throughout the plant body and thus constitutes 80 to 90 per cent by weight of the active plant cell. (2) It serves as one of the crude materials (water plus carbon dioxide) in the process of carbohydrate food manufacture, or *photosynthesis*, by all green plants. It thus furnishes the hydrogen and oxygen of sugars and starches which are later used for food and also takes part in other necessary chemical processes, such as the hydrolysis of the complex foods like carbohydrates, proteids and fats. (3) It makes possible the *maintenance of turgidity* or a hydrostatic pressure within living cells, a condition which is necessary for, or essential to, growth. (4) It supplies transpiration or evaporation—the loss of water through aerial parts—and thus promotes and regulates growth. Water loss by transpiration is a measure of growth and the accumulation of dry matter in our plants. The internal structure and the external form of plants may be profoundly altered by the variations in the water relations either by the soil moisture or by the humidity of the air. A detailed consideration of such alteration would carry us into the province of plant physiology and ecology, but it must be evident that excesses or dearth of water will so affect nutritive or other physiological processes as to produce either disease or death of cells, tissues, organs or entire individuals.

General Effects of a Disturbed Water Relation.—The demands of our crop plants for water are exceedingly variable, and even certain varieties of a species may be much more sensitive to moisture fluctuations than others. A plant may be provided with too much or too little water, or water may not be available at the right time. The health of a plant is affected by the moisture supply of both its soil and air environment, and the amount needed for a normal or thrifty development is influenced by various environmental factors, such as temperature, sunshine and physical properties of the soil. The effects of a water shortage will vary in the case of sudden and acute shortage or in chronic deficiency of water throughout a long period. The first marked response to a pronounced

water shortage is wilting, the drooping of succulent shoots and the rolling of leaves. The loss of water faster than it can be brought up from the root system decreases the turgor of the cells, and the structures, normally tense or rigid, become limp and flaccid. This *physiological* wilting is a common sight on a hot summer day, but the wilted structures again become rigid during the night or when evaporation is retarded, and thus growth proceeds with only temporary checks. Marked water shortage will result in retardation of growth and dwarfing. Moisture deficiency is rarely operating as a single factor, since high temperatures and intense sunshine are frequent accompaniments. A plentiful water supply stimulates growth and results in the production of succulent tissues as contrasted with the firmer structures of plants supplied with moderate amounts of moisture.

Effect of Moisture Deficiency.—If the shortage of moisture is not relieved, drought response in foliage is marked by yellowing, reddening or other discolorations, followed by leaf fall in woody plants. In plants suffering from drought, dead brown areas may appear in the intercostal areas of leaves, in the center of areolæ of these areas, or the leaves may be blighted or burned at the margins or tips. It should be remembered that other factors, such as toxic substances acting internally or externally or intense light and heat, may give rise to somewhat similar symptoms.

Moisture shortage by its interference with nutritive processes may lessen the production and storage of reserve food. Tuber or root crops will remain small, and cereals will produce shriveled grains. Fruits may be spotted, deformed or under normal in size, or they may shrivel and fall prematurely. In woody plants, drought injury may not be evident entirely during the season of low moisture, but the effect may be delayed until the next season, when weak shoots may be formed or twigs or branches die back, producing *staghead* or *dieback*.

The transplanting of herbaceous plants or of nursery trees frequently causes a disturbance in the water relations that may result in either death or a retarded growth. If herbaceous seedlings are grown in the moist atmosphere of a greenhouse, hot bed or cold frame, the cuticle is delicate, the external epidermal wall thin and the tissues in general poorly suited to withstand the rapid transpiration of a dry air. Hence if such plants are suddenly transplanted to the field, they may wilt so rapidly that death will result. If the root system is broken or mutilated, the danger of injury is greatly increased. The reduction of loss in transplanting may be accomplished by: (1) the hardening of plants by gradually subjecting them to conditions which approximate those of the field, rather than by a sudden change; (2) care in preventing the mutilation of the root system; (3) cutting back the top or removing leaves to bring about a balance between transpiration and absorption until the root system can provide the necessary water; or (4) the protection of the transplanted plants

from the direct rays of the sun or from the force of the wind so as to retard transpiration until the plants have become established. Because they are not able to adjust themselves to the new conditions, many delicate plants of the greenhouse, if moved to the dry air of living rooms without being hardened, may wilt, drop their leaves and even succumb.

Some Effects of Excess Moisture.—The injurious effect of water-logged soils has been pointed out (see Chap. VI on Diseases Due to Improper Air Relations), and the importance of soil oxygen in the life of our crop plants emphasized. It has been shown that, in addition to the phenomena of yellowing and decompositions connected with an over-supply of water in the soil, there is an actual decrease in production. Recent attention has been directed to the fact that any effect on the plant resulting from driving out the soil oxygen or the prevention of aeration may be due to either the lack of oxygen or the excess of carbon dioxide which cannot be carried away (Knight, 1924) acting directly on the plant or on the soil organisms. It is significant that water cultures of corn failed to respond to aeration of the nutrient solution.

An abundant water supply produces a type of growth that is more susceptible to the inroads of either bacterial or fungous pathogenes (see Fire Blight) and is more sensitive to extremes of heat or cold. This may be illustrated by the *sun scald* of the potato. When a period of warm rain, which checks evaporation and supplies abundant water, is followed by a period of high temperature and bright sunshine, the leaf tissues which are suffused with water are frequently injured, and a sudden blighting of leaflets or portions of leaflets results. This condition is sometimes mistaken for the invasion of a parasite. The same temperature and illumination would have no injurious effect on tissue having a normal water relation.

The greater delicacy of the cell walls in plant structures provided with an abundance of water and the increased rate of growth frequently result in the rupture of fleshy organs, such as fleshy roots, tubers, stems or fruits.

> All of these phenomena have characteristics in common—that they are initiated only when, after a considerable period of normal development, or still more after a previous dry period, an unusual supply of water is given suddenly (Sorauer and Dorrance, 1914).

This behavior is well illustrated by the cracking of carrots, kohl-rabi, beets, turnips, potatoes and even some herbaceous stems. In the potato, the rupture is frequently internal and then the condition known as "hollow heart" results, in which a central cavity is formed, bordered by brown, oxidized tissue. *Hollow heart* is most frequent in potatoes which have been stimulated to an excessive growth by abundant moisture, as is frequently the case in irrigation farming. The rupturing of nearly

mature, soft-skinned fruits, such as cherries, plums or tomatoes, when a rain follows a rather prolonged dry period is a fairly common phenomenon. These troubles have been proved to result from high sap pressure due to excessive water supply.

The knot-like or pustule-like enlargements on various organs, such as stems, leaves or fruits, produced as a result of the enlargement of groups of cells, are known as *intumescences,* while more extended swollen areas in which similar tissue changes have taken place are characteristic of the disease known as dropsy or *œdema.* A similar trouble of the tomato has been described in some detail by Atkinson (1893), who came to the conclusion that too much water in the soil was one of the causes, although insufficient light and improper temperature relations were favoring factors. Many theories have been proposed to explain the formation of intumescences and similar deviations from the normal development, but these cannot be given a detailed discussion. Suffice it to say for our present purposes that they represent a disturbed nutrition of the cells in which the cell walls remain relatively thin and the cells become distended with cell sap, frequently reaching many times their natural size. It may also be noted that intumescences have been produced experimentally by either chemical or mechanical stimulation (Smith, 1920). The phenomenon of bitten or perforated leaves, in which leaves are very much shredded, torn or perforated by irregular openings, is closely related to the formation of "intumescentia" from the standpoint of origin. The production of enlarged lenticels in potatoes or other underground organs, as the result of excessive moisture, is also similar to the formation of intumescences. The so-called "tan disease" may also be mentioned in this connection. The bark of either roots or aerial portions becomes more or less swollen in either localized or extended patches, and the outermost cork layers break or peel away. The surface beneath the blisters may show a whitish granular or even woolly appearance due to large numbers of loosely arranged cells, many of which may become more or less elongated. These loosely arranged cells die and then, under dry conditions, appear as a dry, reddish-yellow, brownish-yellow or brown powder which may easily be brushed off. This condition may be induced on the apple by severe heading back to prevent transpiration at a time when root absorption is active. A somewhat similar type of cell formation is responsible for the "woolly streaks" in the cores of certain apples, and the tendency to form such streaks may be especially pronounced in particular varieties.

The fall of leaves, the shedding of blossoms, the dropping of fruits or the casting of twigs may sometimes result from a disturbed water relation, either an excessive supply, a shortage or abrupt fluctuations, although various other nutritional disturbances may play a prominent part. Such troubles as June drop of fruits, failure of fruits, like grapes, to set or,

later, their shelling, the blossom drop of tomatoes and the shedding of cotton squares or bolls may be mentioned in this connection. Although there are many factors that play a part in reducing the set of fruit, excessive rains and prolonged humid conditions at blossoming time play an important rôle. Aside from the indirect effects of such conditions on the nutrition of the plant, rain washes pollen down to the ground, restricts either wind or insect pollination, causes pollen grains to burst and also washes away stigmatic secretions which promote the germination of the pollen grains.

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BITTER PIT

Of the several spot diseases of the apple, the widespread trouble characterized by the appearance of circular or slightly irregular depressed spots on the surface of the fruit and also by internal necrotic areas, and known most generally as bitter pit, is the most important. At various times, it has been described under a number of other common names, such as fruit spot, apple brown spot, spotted apples, Baldwin spot and fruit pit by English and American writers, as "Stippen," "Stippflecke," "Stippigwerden" or "Stippigfleckigkeit" by the Germans and as "liège" or "points bruns de la chair" by the French.

Spot Diseases in General.—Bitter pit has not always been clearly differentiated from the other non-parasitic spot diseases of the apple. The most important of this group of troubles and their distinguishing characters are as follows:

1. *Bitter Pit.*—Superficial, circular or subcircular, depressed spots and internal necrotic areas.

2. *Spot Necrosis or Drought Spot.*—Large, irregular, water-soaked spots generally located toward the calyx end, which later become depressed and show a shallow layer of dead brown tissue below the spot.

3. *Cork* and its modifications, known under such names as blister, malformation, dry rot, York spot, punky disease, crinkle and hollow apple. Internal dry, punky patches of tissue, much more extensive than in bitter pit and with or without external malformations in the form of depressions or ridges. Hollows may be formed in larger dry-rot areas by shrinkage of affected tissue. The disease may appear with or without reduction in size, depending on severity. In blister, the surface is first covered with brown, more or less circular, raised spots, which may later crack and scale off, leaving the surface rough.

4. *Jonathan Spot.*—Circular depressed spots, minute to ¼ inch or slightly more in diameter, always centering at lenticels, with a shallow area of necrotic tissue but no internal necrotic areas as in bitter pit. Common on the Jonathan but not confined to that variety.

5. *Jonathan Freckle.*—Circular areas of discolored tissue up to ¼ inch in diameter, only skin deep and not becoming depressed. Appears only in storage.

6. *Scald.*—Brownish discolorations of the skin involving rather extended areas, especially on the lighter cheek, sometimes followed later by internal discolorations of the pulp (common scald) or subcircular or irregular elongated areas of brown tissue having much the appearance of a fungous rot (soft scald). Appears only in storage (see more complete account, p. 125).

7. *Stigmonose.*—This name is applied to the abnormal fruit conditions resulting from the feeding or egg-laying punctures of certain insects, by which the fruit is spotted, pitted, malformed and sometimes reduced in size. Due to rosy aphis, red bugs, thrips, leaf hoppers, etc.

In much of the earlier American literature, bitter pit and the parasitic fruit spot (*Phoma pomi* Pass.) were confused. This fungous disease of the eastern states was first clearly differentiated from bitter pit by Brooks (1908).

History and Geographic Distribution of Bitter Pit.—This disease was first recognized in Germany in 1869, and the name "Stippen" has been used more frequently than any other, being given to the trouble by Wortman (1892). It was recorded as fruit spot by Jones, as early as 1891 in Vermont, and later as brown spot and Baldwin spot. It was first referred to as "spotted apples" by Crawford in his study of apple troubles in South Australia, published in 1886. The earliest report from

England was in 1905, the trouble being described as the apple brown spot. Bitter pit, the name now in most general use, was first applied by Cobb (1895), who studied the disease in New South Wales. The disease was the subject of study by Evans in South Africa in 1909, but the most extensive studies were begun in Australia in 1911 as the final outcome of an agitation that was started by the National Fruit Growers Conference of Australasia in 1908. By the combined support of the Federal government and the several states of the Commonwealth, the investigation of the bitter-pit problem was assigned to McAlpine, and five extensive reports (1911–1912 to 1915–1916) were issued. Following the first reports of the disease in the United States, it received frequent mention by experiment-station workers, but little information was contributed beyond the description of symptoms and effects, the recognition of its non-parasitic origin and theorizing as to possible cause. Delay in emphasizing the investigation of bitter pit in the United States was due partly to the presence of numerous parasitic diseases which absorbed the energies of experiment-station workers, and partly because in the earlier years there was not so great a demand for high-grade fruit. The development of the irrigated orchards of the West and the emphasis on the production of extra-fancy fruit for eastern markets and foreign shipment have brought the bitter-pit problem to the attention of our orchardists and have been a stimulus to more recent work. The problem was given special attention by the Federal Office of Fruit Disease Investigations, and several reports have been issued by Brooks and Fisher (1916, 1918) based largely on the work of Fisher in the important Wenatchee district of Washington. These reports have been valuable contributions to our knowledge of the spot diseases in general and have given new light on the etiology and control of bitter pit.

Although there is no available proof of the fact, it seems probable that bitter pit has affected the apple from the time when it began to be generally cultivated. This may be inferred from the physiological nature of the trouble. It is certain that the disease was recognized in Europe long before any definite name was ever applied to it. At the present time, bitter pit is recognized as a disease of apples wherever they are grown. It is not, however, prevalent to the same extent in all commercial apple districts but seems to be more frequent and severe in those localities in which there are the greatest chances for a disturbed water relation of the growing crop. Bitter pit, therefore, is of most concern in irrigated districts where the normal conditions are semiarid and where the water supply is a variable and fluctuating factor. Even under natural conditions, certain localities, from their normal soil and climatic relations, may favor the trouble. It is sometimes stated that growing the apple where nature never intended it to grow has increased the amount of bitter pit. This will probably explain why it has been most severe in certain parts of America and Australia.

Symptoms and Effects.—Bitter pit is a disease that is confined entirely to the fruit. The trouble may be very severe on trees which appear to be in prime health if judged by vigor of growth and luxuriance of foliage. The disease does not make its appearance until after the fruit is half grown and generally is not evident until the fruit is approaching maturity. In many cases, it does not appear on the fruit previous to harvesting but becomes evident later, generally during the early part of the storage period.

The first external evidence of the disease is the appearance of slightly discolored spots on the skin of the fruit—darker red in red fruits and a deeper green on the yellow-skinned fruits. At first these spots are not depressed, but soon they become more or less sunken and assume the

character of typical bitter-pit lesions. Such spots are more or less circular and vary in size from minute specks to others ¼ inch or more in diameter and appear like dents in the skin. They are not localized on any particular part of the fruit but are likely to be most numerous toward the calyx end and even in the most severe cases are absent from a small area around the stem. Two or more pits standing adjacent may be confluent and so give rise to larger and more irregular lesions. The coloration of the skin as noted for the young spots persists for some time in the pits, but finally the depressions become brown due to the death of both surface cells and

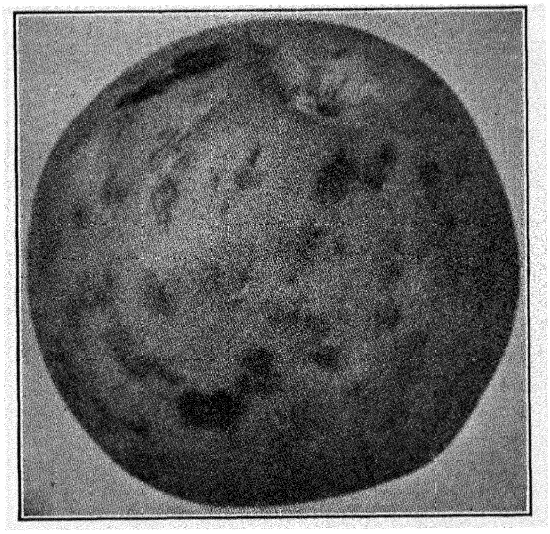

FIG. 38.—Bitter pit of apple. (*After McAlpine.*)

underlying pulp tissue. The skin lining the pits remains unbroken throughout the course of the disease.

When an affected apple is cut in two, it is generally found to show internal groups or masses of brown necrotic pulp cells which show no connection with the external pits and the necrotic tissue just beneath them. The internal necrotic areas are generally most numerous in the peripheral portion of the pulp, but they may occur at any point outside the core wall. It is frequently noted that the fruits which show no external marks of the disease are already affected internally. This can readily be demonstrated by cutting into apparently normal fruits in lots that show a good percentage of evident pitting. Thus we may find abundant surface pitting and few internal necrotic spots, internal lesions with no external pits, or the lesions may be abundant both on the surface and in the interior.

The pulp or flesh directly beneath a pit as well as the discolored spots in the interior consist of a mass of dead brown cells, dry and more or less corky or spongy in character. At first the dead tissue is a light

brown, but later it becomes a darker brown. The tissue of the lesions generally has either a slight or a very pronounced bitter taste, so that this character coupled with the pit-like depressions makes the name "bitter pit" especially appropriate. Although other non-parasitic spot diseases resemble the bitter pit and certain parasitic spots like the New England fruit spot appear somewhat similar, when one really becomes familiar with bitter pit, it is not likely to be mistaken for any of the other troubles.

Bitter pit may vary greatly in its severity and the damage which it causes. Apples may show only a few small spots, or the lesions may be as numerous as the pits on the face of a person who has suffered a severe

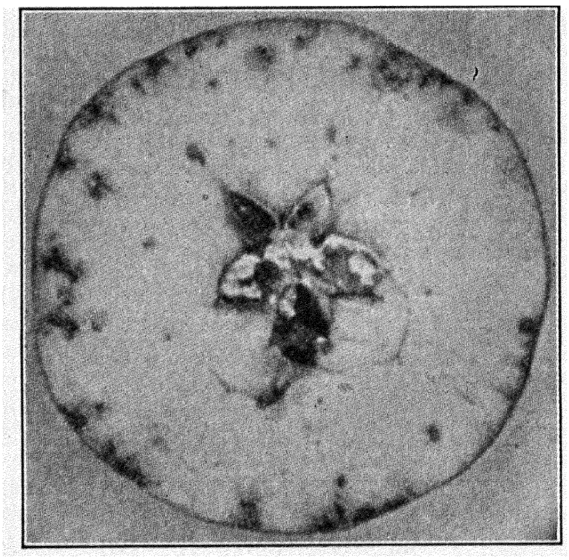

FIG. 39.—Section of an apple showing bitter pit and also moldy core. (*After McAlpine.*)

attack of smallpox. Such names as "measles" or "smallpox" have found local use instead of bitter pit, but they have never been generally adopted. The affected apples are not destroyed, but they are of poor quality and also inferior in appearance. The disease is of special concern from its effect upon grade or pack, especially when so much attention is being paid to the production of extra-fancy fruit. A crop showing bitter pit at picking time is likely to suffer deterioration during storage even though carefully sorted and should not command the price of the best fruit. Rot-producing fungi sometimes establish themselves in old bitter-pit lesions and thus invade the fruit when normal fruits would escape. No accurate estimates of the actual losses from bitter pit are available, but it is certain that the disease is of world-wide importance and is taking a very considerable annual toll.

Etiology.—Although bitter pit bears certain resemblances to a parasitic trouble, it has been definitely proved that no organism is asso-

ciated with it in a causal capacity. It is now very generally conceded that it is a non-parasitic malady associated with a *disturbed water relation.* In the light of present information, it is a disease of definite and pronounced symptoms, just as truly a disease as though caused by a specific pathogene; hence the statement of Lewis (1915) may be viewed with surprise that "there is some question whether it is a true disease, it seemingly having more of the characteristics of a physiological breakdown."

Even after bacteria, fungi, insects, mechanical injuries, degeneration from old age, varietal peculiarity and unfavorable grafting were excluded as causes of bitter pit, some untenable theories persisted. The one which received the most emphasis was the poison theory of White (1911)

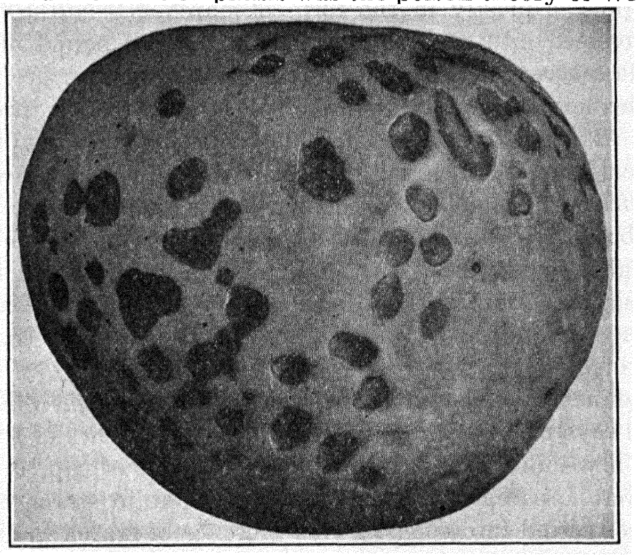

FIG. 40.—Late stage of bitter pit on Rhode Island Greening apple. (*After Brooks and Fisher.*)

and Ewart (1912 and later). It was at first suggested that the absorption of arsenical compounds or other spray materials through the skin of the apple was responsible for the killing of localized groups of cells. This was modified later to the view that the toxic substances responsible for the killing of the cells were absorbed by the root system and distributed through natural channels. This poison theory was held to rather tenaciously by its advocates, but it has not been substantiated by other workers (McAlpine and coworkers, 1914–1915; Crabill and Thomas, 1916).

Even with the agreement that bitter pit is due to a disturbed water relation, there has been no unanimity as to the exact way in which the disturbance operates. While this is a difficult thing to prove, the possible origin can be better understood with a clear notion as to the structure and functions of the normal fruit. The fruit, as well as the

leaves, has its own supply of fibrovascular bundles, along which water and mineral substance pass. These water-conducting vessels enter through the fruit pedicel and are distributed throughout the pulp, but a very large number terminate in the peripheral portion of the fruit. The pulp cells occupy the spaces between the vascular network, and the whole apple is protected by a suberized and nearly impervious epidermis, interrupted only by lenticels through which there may be entrance or exodus of gases and through which aqueous vapor may escape in the process of transpiration. Water and mineral substances taken from the soil, and carbohydrates manufactured by the leaves, are carried into the apple and distributed to the pulp cells. The carbohydrate in the growing apple is mostly in the form of starch, but as the fruit ripens this is converted into sugar. During the growing period of the fruit, transpiration is very active, and large amounts of water are passed out through the lenticels, just as water is lost from leaves through their stomata. Transpiration is not simple evaporation of moisture but is a physiological process which will be affected by supply of moisture to the root system, air humidity, air movements, temperatures, light, etc. There is abundant evidence that the tissues of growing fruits may suffer from drought injury (drought spot or spot necrosis) when the foliage shows no signs of injury. The higher sap pressure of the leaves makes possible the withdrawal of water from the fruits under conditions of water shortage.

An examination of the groups of dead cells in bitter pit will show that they are always closely connected with certain branches of the vascular bundles. The individual cells are brown, devoid of sap, more or less collapsed but apparently not ruptured and contain starch grains that were not converted into sugar. This presence of starch in the affected cells has been taken to indicate that the changes which initiated the injury occurred before the ripening processes were completed. It has also been definitely proved that bitter-pit lesions, which first make their appearance after the fruit is in storage, are always located and the injury started while the fruit is still on the tree. It is interesting to note that in drought injury to the foliage of a plant, the first tissues to suffer are either at the margin of the leaf or at the termini of veinlets in the areolæ of the leaf venation—in other words, at the ends of the water-conducting vessels. This behavior is simulated in the location of the lesions in the bitter pit, the surface pits being at the periphery of the water-conducting system.

Five theories[1] have been advanced to explain the killing and drying out of the affected cells as the result of a disturbed water relation:

[1] It has been suggested by Kidd and West that bitter pit may be due to localized poisoning from the temporary accumulation of excesses of carbon dioxide. This is based on their studies of brown heart (see reference under Scald).

1. *The Ruptured-cell Theory.*—"When there is an extra rush of sap following on dry conditions, the rapidly swelling pulp cells at the external boundary burst the vascular network at localized spots, and the sap pressure, which is sufficient to rupture the enveloping network, also bursts the thin-walled pulp cells at these particular spots, and death of the cells ensues. Briefly, it may be stated that rapid alternations between dry and moist conditions, combined with fluctuating temperatures during the growing stages of the fruit, is the exciting cause of bitter pit" (McAlpine, 1921).

Such a condition might be furnished, for example, by active absorption by the root system in a warm soil, while the transpiration is checked by low night temperatures.

2. *The Crushed-cell Theory.*—"The affected cells have been killed by being crushed by neighboring cells having higher osmotic pressure due to their higher proportion of sugar" (Herbert, 1921). According to this theory if "there is a sudden rush of sap into the apple, the cells which had their starch converted into sugar will swell to a greater extent and more rapidly than those cells which are still supplied with starch. The rapid distension of the cells is resisted on the outside by the skin. Their force of expansion results in the crushing of those cells whose starch transformation is backward."

It is the belief of the exponents of this theory that it is in accord with certain recorded facts which are opposed to the ruptured-cell theory as follows: (*a*) the sunken character of the spots; (*b*) the unbroken skin over the bitter-pit lesions; (*c*) the frequent continuation of vascular bundles through a lesion to supply normal tissue beyond; (*d*) the presence of starch in the pit cells; and (*e*) the great tensile strength of cellulose which would make bursting of the cells unlikely. On the basis of this theory, it is suggested that immunity or resistance to bitter pit might be due to a uniform transformation of starch into sugar throughout the fruit.

3. *The Starved-cell Theory.*—As a result of water shortage, certain groups of cells fail to receive sufficient mineral food and thus die from starvation. This might explain the disease during very dry seasons, but it is not in keeping with the occurrence of the trouble in severe form on vigorous trees well supplied with moisture.

4. *The Concentrated Cell-sap Theory.*—"In bitter-pit tissue the pulp cells have collapsed, and the brown flecks in the flesh contain much less water than the neighboring healthy tissue. Owing to this loss of water, the acids and other constituents of the cell sap have become concentrated, and the amount of concentration reaches a point where death ensues. The concentration of the cell sap is, therefore, in all probability, the immediate cause, acting directly upon the protoplasm, and it must not be forgotten that this concentration is increased by an insufficient supply of water as well as by excessive transpiration" (McAlpine, 1914–1915).

This theory was later abandoned by McAlpine, who placed first emphasis on the ruptured-cell theory (1915–1916).

5. *The Plasmolyzed-cell Theory.*—This is really based on a different interpretation of the conditions outlined under the "crushed-cell" theory. According to Carne (1927), the osmotic pressure in groups of cells containing starch is greater than that in surrounding cells containing sugar; hence water is withdrawn from the former, resulting in their plasmolysis and death. It is contended that the trouble originates in storage as well as on the trees.

Predisposing Factors.—Acceptance or rejection of a theory does not alter the fact that certain soil or climate factors or cultural practices favor or promote the disease. Many observations have been made and various opinions are more or less conflicting. Some of the statements are: Bitter pit is favored by (1) soils of poor physical or mechanical condition, especially those that are low in humus and have a poor water-holding capacity; (2) alternating wet and dry weather or very dry conditions followed by heavy rainfall, especially if this fluctuation comes relatively late in the season; (3) light irrigation during the early part of the season and late heavy irrigation; (4) heavy irrigation throughout the season, but less than by the variable light and heavy irrigation; (5) light irrigation throughout the season more than by heavy irrigation followed by light; (6) conditions which bring about the production of a light crop of large-sized fruit or of oversized fruit in general; (7) heavy pruning or a system of pruning which throws the fruit production onto the main limbs rather than on well-distributed laterals; (8) fluctuating temperatures and humidity near the ripening period; (9) excessive transpiration or climatic conditions which cause a water loss out of keeping with the available supply. When conditions, whether natural or artificial, are such as to promote a uniform growth from early spring to the ripening period, the chances of bitter pit will be reduced to a minimum.

In stored fruit, the disease will not appear unless it was initiated during the growing period, but if the fruit is already affected, the disease will progress most rapidly if the storage temperature is relatively high and will also be favored by fluctuations in humidity and temperature. In other words, it will be favored by conditions which promote cell activity and retarded by conditions which delay or retard maturing or aging of the tissues, hence by low temperatures. It is also stated that in some cases at least, the disease is worse on early-picked fruit than on well-matured or late-picked fruit (Smith, 1926; Carne, 1927 and 1928*b*). Because of this relation these authorities recommend later picking as a preventive. In recent studies, Heinicke (1921) has presented data to show: (1) that the lateral fruits of a cluster are more likely to develop bitter pit than the central fruits; (2) that fruits on basal spurs are more

affected than those toward the tip of a branch; (3) that apples on weak wood are more subject to the disease than those on strong branches; (4) that there is a relation between the amount of bitter pit at harvest and the seed content of the apple, affected fruits averaging lower seed content and more poor seeds; and (5) that new pits which first appear in storage are more abundant on the large many-seeded sides of unsymmetrical fruits.

Varietal Susceptibility.—The apple is the crop most seriously affected by bitter pit, but it is recognized as a disease of pears and quinces. The different varieties of apples appear to show different degrees of susceptibility. Some are recognized as uniformly and severely affected, others as medium in susceptibility, while others are only slightly susceptible or almost immune. According to some reports, varieties very susceptible in one locality are reported less susceptible in another region. In North America the Baldwin is conceded to be a very susceptible, if not the most susceptible, variety, but in some states of Australia it has been reported as medium in susceptibility. The Cleopatra (Ortley) and Northern Spy pit very badly throughout all the states of Australia, and the latter variety is listed as among the most susceptible in America. The Stayman is a very susceptible variety, especially in the Pacific Northwest. Probably the great majority of our best commercial apples are either very susceptible or moderately susceptible to the disease, so that little relief can be expected from the selection of resistant varieties, but hope has been offered that pitproof varieties of high commercial value may be obtained by the crossing and selection of liable and non-liable varieties.

Control.—The effort of the orchardist must be directed, first, to providing growing conditions which will reduce the incidence of the disease to a minimum; second, in case the trouble has developed, to the retardation of its progress in storage or in transit to market. Experience will soon tell whether the disease is sufficiently frequent and serious in any environment to call for careful attention to control measures. It seems to be true that many of our best sections for commercial production are especially favorable for the production of bitter pit; hence in such localities the grower must constantly be on guard and follow those practices which are known to be of value. The complex character of the physiological disturbances which initiate bitter pit make it at once evident that no single practice will give adequate protection. The following practices should be the guide in so far as they can be put into operation: (1) Try to increase the fertility of weak soils by the liberal use of manure or by the use of a cover crop suited to the locality; (2) follow cultural practices that will tend to conserve moisture or that will insure as even a distribution throughout the season as possible and that keep the soil well aerated and thus provide favorable conditions for the normal root activities; (3) avoid crowding of trees and overbearing of fruit

and also guard against light settings of fruit coupled with extra-vigorous vegetative development; (4) practice a type of pruning that will insure a crop that is evenly distributed over the tree, with the retention of the laterals for the bearing wood with minimum fruiting on the main axes; (5) avoid heavy pruning, as this will throw the root system and top out of balance and consequently may modify transpiration and the general water relation of the tree; (6) follow a well-planned system of thinning of fruit, not too great or too little, as undersized or oversized fruits are more prone to the disease than those of moderate size; (7) in irrigation farming, give special attention to the amount of water used and to the time of application, avoiding overirrigation or underirrigation and especially heavy, late irrigation. The relation of irrigation to bitter pit is summarized by Brooks and Fisher (1918) as follows:

> Heavy irrigation throughout the season has given less of the disease than medium irrigation followed by heavy, and light irrigation throughout the season has resulted in more bitter pit than heavy irrigation followed by light. Heavy irrigation the first half of the season caused the trees to develop a more luxuriant foliage and probably produced a lower concentration of the cell sap in the apples, both of which facts would tend to make the fruit less susceptible to the forcing effects of late irrigation. The amount of irrigation in August and September has apparently largely determined the amount of the disease. The results as a whole point to the harmful effects of heavy late irrigation regardless of the size of the fruit.

(8) Avoid too early picking (Carne, 1927 and 1928*b*).

With the recognition that bitter pit will become more pronounced during the storage period provided the fruit was affected at the time of harvest, two courses are open to prevent loss: (1) early marketing to bring the fruit to the consumer before the disease has had time to develop to serious proportions; or (2) retention of the fruit under transit or storage conditions that will prevent any further advance of the trouble. At temperatures of 30 to 32°F. cell activity is reduced to a minimum, and consequently bitter pit can make little further progress, but fluctuations beyond these limits should be prevented. By the proper use of refrigeration, it is possible to send apples around the world with little danger of loss from bitter pit.

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BLOSSOM-END ROT OF TOMATOES

The tomato is affected by a serious disease characterized by a dry rot of the blossom ends of either green or maturing fruits. In some of the earlier studies of the trouble, it was described as the rot, black rot, fruit rot, point rot, dry rot or dry-weather rot, but blossom-end rot has been the name in most general use in recent times. Blossom-end rot is preferable to any of the other names, since it gives the best expression of the nature of the trouble.

History and Geographic Distribution.—The first careful study of this tomato disease was published by Galloway (1888), who stated that at that time specimens had been received from nearly all parts of the United States. From present-day knowledge of the disease, it seems probable that it must have been prevalent ever since the modern varieties of tomatoes have been grown; at least it seems now to be of world-wide occurrence. Galloway found two fungi associated with the disease, a species of Macrosporium and a Fusarium, but considered the former as the first cause of the rot and the latter as a secondary form which assisted in the disorganization of the fruit. Following this time, little progress was made in our knowledge of the disease until the work of Jones and Grout (1897). Up to this time, the conclusions of Galloway had been quite generally accepted, and spraying tests had been carried out for the control of the disease, but Jones and Grout showed that the fungus which frequently accompanied the disease was not a Macrosporium but *Alternaria fasciculata*, a widely distributed saprophyte, which occurred on many kinds of decaying vegetable matter and that it would not induce the disease when inoculated under the skin of green tomatoes.

Since the disease did not seem to be due to a specific fungous pathogene, it was but natural that investigators should turn to bacteria as the causal agents. Prillieux and Delacroix had made such a suggestion as early as 1894, but in 1900 Earle reported that "as the result of studies extending over the past 3 years, it has been quite certainly proved that the disease is bacterial not fungal, being caused by the growth of an undetermined species of Bacillus." Stuart (1900) in Indiana had also attributed the disease to a similar bacterial parasite. The bacterial origin of the disease was supported by the investigations of Elizabeth Smith (1907), but she also concluded that a similar rot could be caused by *Fusarium solani* working independent of any other organism. After working with the disease for 16 years, Stone (1911) stated that the work of Miss Smith had been repeatedly verified in his laboratory and greenhouse. The fungous origin of the disease was again defended by Oven (1905), who attributed it to *Fusarium erubescens*. Even as recently as 1913, Groenewege came to the conclusion that the disease is due to a bacterial parasite which he described as new under the name of *Phytobacter lycopersicum*. His conclusions, like those of previous workers, were based on the results of inoculations. In a careful perusal of the various studies which claim a bacterial origin of the disease, the critical reader will note that the conclusions were drawn from very incomplete evidences.

During the progress of these earlier investigations, some scientists had failed to accept the conclusions as to the parasitism of any of the associated organisms, holding to the view that neither bacteria nor fungi are the causal agents. This view was supported by the careful studies of Stuckey (1901), who determined that the blossom-end rot is a non-infectious disease, which "can be controlled, if not entirely prevented, by keeping an abundant supply of water in the soil." These conclusions were corrob-

orated by the careful work of Brooks (1914), who contributed additional data concerning the etiology of the disease. It is of interest to note that later studies showed the transmission of either resistance or susceptibility to blossom-end rot from parent to progeny (Stuckey, 1916). As a result of these later investigations, it seems to be quite generally accepted that the disease is of a non-parasitic character. It has, however, been suggested (Reynolds, 1918) that the disease "is due to an organism probably ultramicroscopic, which infects at pollination time only and causes the rotting area to enlarge until such time as the physiological processes of maturing have changed the composition of the fruit so much that further invading of the tomato tissue is impossible for the organism." This must, however, be regarded purely in the light of a hypothesis.

Symptoms and Effects.—The first evidence of the disease is generally the appearance of a dark-green, water-soaked area at the base of, or

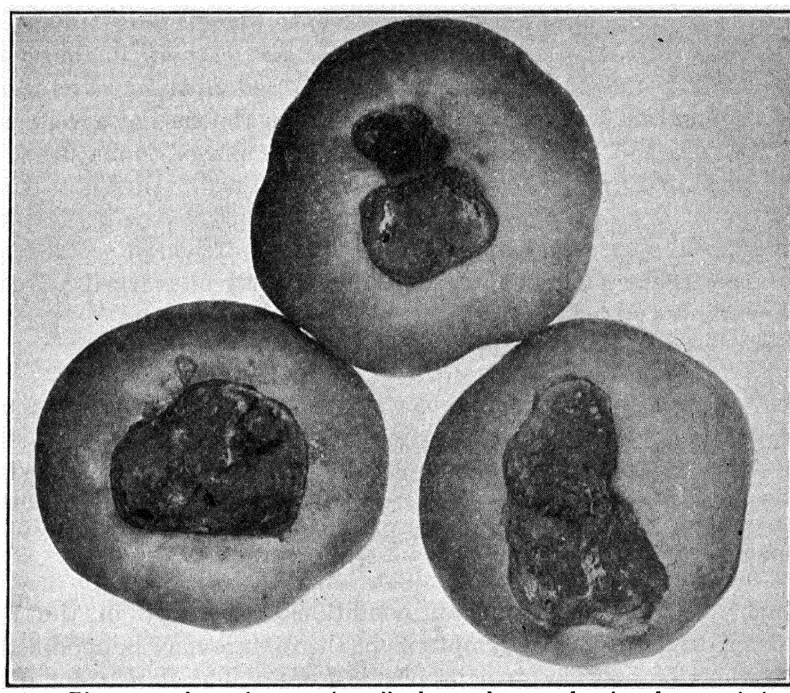

FIG. 41.—Blossom-end rot of tomato in well-advanced stage, showing characteristic appearance. (*After Stuckey.*)

surrounding, the remains of the style. This watery spot is quite similar in appearance to water-core spots in apples. The initial area may be just around the style or somewhat removed from it, or it may cover the whole blossom face of the fruit at the same time. This watery area is confined to the portion immediately under the skin and does not involve the tissues to any great depth even after the lesion has reached some size. The affected tissues cease to grow, the dark-green, water-soaked area turns to a lead color or brown and the spot becomes flattened or even sunken, while its color may be deepened to almost black, although

there is much variation in the depth of color under different conditions. The trouble may appear on very young fruits, although it is frequently not much in evidence until they are an inch or more in diameter. The lesion may increase in size with the growth of the fruit, but it does not advance any after the fruit has turned red. The affected area is generally nearly circular in outline and may occupy only a small portion of the blossom end, or a maximum of nearly one-half the total surface may be involved. The discolored area may be uniform throughout and clearly marked off from the surrounding normal tissue, but the advancing edge is frequently marked by a narrow darker zone. In some cases, a slight concentric zonation may appear within the lesion, due to varying shades of brown. It is a common thing for the ripening of the diseased fruits to begin at the periphery of a lesion and advance toward the stem end.

The affected tissue is firm and more or less leathery in the typical development of the disease and after it has dried down looks as though it had been seared with a hot iron. Except in the case of a very early and severe onset of the disease, the fruit will be carried to maturity, but the ripening processes will be more or less hastened. In slight attacks which appear late in the growth of the fruit, there may be no flattening of the affected area, which appears more like a scorched or scalded spot. In the most slight infections, the blossom end may be marked by only a slight scurfiness under the skin.

According to Brooks (1914):

> The first effects of the disease are not always superficial. Fruit that appears entirely normal from an external view often has the tissue of several, or sometimes all, of its placentæ collapsed and blackened in the parts nearest the blossom. In some cases, this internal condition is accompanied by a very inconspicuous depression of the surface tissue above it or by a small water-soaked area on the surrounding surface.

Under certain environmental conditions, especially in the more humid regions, the normal symptoms of the disease may be modified by the entrance of bacteria or saprophytic fungi. The dark color of the lesion may be intensified by the presence of a sooty fungus, and the superficial development of the organism may give a dark velvety appearance to the affected portion. It was this condition which suggested the early name of "black rot" which was applied to the disease. The lesions may sometimes be invaded by bacteria, and a sticky exudate may be in evidence under moist conditions, somewhat similar in appearance to the bacterial exudate so characteristic of fire blight. It was this condition which led Earle (1900) to make his studies by which he decided that the blossom-end rot was a specific bacterial disease. Various bacteria or saprophytic fungi may gain an entrance through the affected tissues and complete the destruction of the maturing or ripe fruits.

Even slightly affected fruits are ruined for market purposes, since the trade demands freedom from blemishes. The affected fruits are injured for home consumption only, to the extent that the tissues are blackened or rotted, since the balance of fruit is suitable for table use. The losses from blossom-end rot may be very slight, while in extreme cases practically an entire crop may be ruined. The disease may appear with equal severity in the field or under glass.

Etiology.—The blossom-end rot of the tomato is a non-infectious disease due to a physiological derangement which results in the death of the protoplasm of the cells of localized areas within the growing fruits,

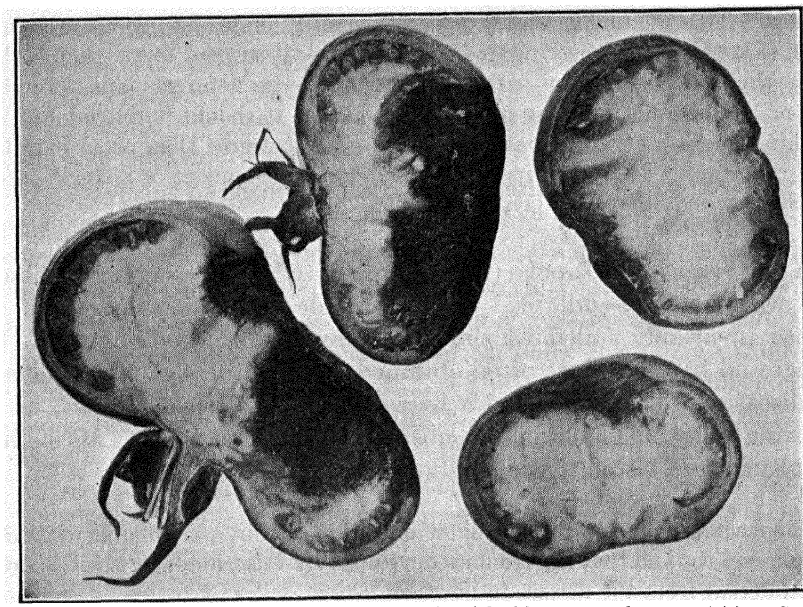

FIG. 42.—Cross-sections of tomatoes affected with blossom-end rot. (*After Stuckey.*)

followed by collapse, drying and discolorations to produce a condition quite properly designated as dry rot. Three periods may be recognized in which different ideas concerning the nature of the disease prevailed: (1) It was believed to be of fungous origin; (2) it was attributed to a bacterial parasite, and proofs were offered which seemed to be adequate to the workers of the time; (3) the present-day concept of the trouble as a non-parasitic disease induced by the operation of environmental factors. In the light of present information, it seems strange that by cultures and experimental tests, able scientists should have convinced themselves that the disease was due to either fungi or bacteria.

The first really conclusive evidence in support of our present concept of the disease was afforded by the work of Stuckey and Temple (1911). By means of cultures, certain bacteria were isolated from typical blos-

som-end rot lesions, but in no case was it possible to produce the disease in typical form, by inoculations, although some rotting occurred in certain cases. Inoculations made by introducing diseased tissue from rotten fruits into healthy fruit or by the use of bacterial exudate also failed to reproduce the disease. To test still further the possible part of a pathogene, the decayed areas from five fruits

> . . . were cut out and ground in a mortar with sterile sharp sand, until the rotted material was cut fine. This mixture of sand and rotten tomato was used to inoculate fifty fruits. The inoculations were made by taking the material on a spatula and rubbing the tip of the fruit until the skin was considerably abraded. The treatment was considered capable of producing more injuries to the skin than could ever occur in nature; also, it seemed to us, that if there was in the rotten areas any pathogenic organism—be it fungus, anaerobic bacteria or bacteria not growing on artificial media—it should be introduced into the skin and flesh of the tomato in a much greater degree than could ever take place in nature. The fruit ripened without the development of a single case of rot (*Stuckey and Temple*, 1911).

A few years later, Brooks (1914) made a study of the organisms associated with blossom-end rot. Cultures made from early stages of the disease invariably failed to show the presence of any organism, but "fungi and bacteria were often obtained from cultures of later stages of the disease." These included a bacterium and a species of each of the following fungi: Penicillium, Macrosporium and Fusarium. All of these organisms were tested by inoculations.

> The results showed that Penicillium and the bacterium were unable to produce blossom-end rot and that the troubles developed by Fusarium and Macrosporium were different from the disease in question.

The possible relation of an enzyme or virus to the disease was tested by making hypodermic injections of filtered extracts of diseased tissue into normal fruits, but negative results were obtained in all cases. This would seem to discredit the theory later presented by Reynolds (1918) of an ultramicroscopic organism.

Of the various environmental factors which may have an effect upon the incidence of the disease, the water relation is the most important. As early as 1910, Selby observed that the rot in greenhouse tomatoes could be checked by a thorough watering, and various other writers agree that the disease was most prevalent on light soils during hot, dry weather. The effect of soil moisture was studied by Stuckey (1911), and on the basis of field tests in which plots were subjected to different moisture conditions it was shown that the "rot on the irrigated plots was very much lower than that on any of the others; and the more thor-

oughly the irrigation was done the less the rot." In later studies by Brooks (1914), the disease was

> . . . produced on vigorous plants by a sudden decrease in the available water supply, but other factors than vigor and dryness have been shown to be of importance in determining the occurrence of the trouble. It has resulted from the wilting of the plants only when their photosynthetic and metabolic processes were in great activity. It has been produced much more readily and uniformly by an excessive water supply than by a scant or intermittent one. Plants receiving a moderate supply of water have developed far less rot than either lightly or heavily watered ones.

In this connection, it is of interest to note that blossom-end rot is rarely serious in certain regions in the Pacific Northwest in which the soil moisture is moderate and gradually sinks to a very low level during the growing season. Under such conditions, plants may suffer from water shortage with little evidence of blossom-end rot. From the experiences in other sections or with tomatoes grown under irrigation in the open or under glass, it seems that water shortage after a period of plenty is more favorable for the disease than a chronic water shortage. The influence of heavy irrigation in the development of the disease is believed to be "due to the development of harmful humic and ammonium compounds at the expense of the nitrate content of the soil." With this interpretation of the results, the heavily watered plants might be considered as growing under conditions somewhat akin to drought, the large amount of toxic substance present increasing the water requirements.

Effects with fertilizers are somewhat conflicting. Stuckey and Temple (1911) state that "there is nothing in the use of nitrate of soda or stable manure to increase the tendency to rot," while Brooks (1914), working under greenhouse conditions, states that

> Increasing the amount of manure increased the rot in all cases. The contrast was greater on a sandy loam than on clay. It is evident that applying the amount of manure often recommended for greenhouse tomatoes (manure to make one-third or one-half the bulk of the soil) may greatly increase the disease and that heavy applications are still more serious.

It was also found that, with liberally watered plants in the greenhouse, potassium chloride increased the disease, while lime and sodium nitrate decreased it, but these relations did not hold true under field conditions, when plants were subjected to drought. In this case, potash did not increase the disease, lime had little tendency to decrease it, while nitrogenous fertilizers favored its development. Contrary to some of the general beliefs, experiments have shown an increase in the disease as a result of staking (Stuckey, 1911), with 68.4 per cent of rot on staked and 20.7 per cent on unstaked plants. This has been interpreted as due

to the more rapid drying of the soil in the staked than in the unstaked plots. It has also been shown that the disease may be increased in amount by raising the soil temperature, and this would suggest that the disease should be less in those localities which have relatively low soil temperatures.

Varietal Relations.—In the tests which have been made, no varieties of our common tomato (*Lycopersicum esculentum*) have proved immune to blossom-end rot, but marked variations in susceptibility have been noted. The cherry tomato (*L. cerasiforme*), the pear tomato (*L. pyriforme*) and the currant tomato (*L. pimpinellifolium*) have been reported as immune. The variation in susceptibility is illustrated by the tests of 26 varieties reported by Stuckey and Temple (1911), ranging from 4.4 per cent of affected fruits in Gold Ball, a small, worthless variety, to 62 per cent in Dwarf Stone. It is encouraging to note that such a valuable variety as the Earliana is among the more resistant varieties. Bonny Best was reported free from the disease during a single test (Brooks, 1914). It is a little difficult to determine comparative resistance under field conditions, since date of fruiting varies and all varieties may not be subjected to the causal conditions at the critical stage in their growth. Apparent resistance may therefore be due to heavy fruiting before the onset of drought or to late fruiting after the trying conditions have been passed. It is the belief that certain physiological characters or structures which make for or against susceptibility are transmitted from parent to offspring. This is based on the results obtained with crosses between the cherry tomato and the common garden form, which have shown high resistance in both the first- and second-generation progeny. It still remains for the plant breeder to develop immune varieties that will be of good size and of high commercial quality.

Prevention.—The early belief that blossom-end rot was due either to fungi or bacteria naturally led to tests of spraying as a method of control. If spraying is of any value, it must be due to control of insect pests or to improvement in the general physiological tone of the plant, possibly through modification of transpiration. The results have been somewhat conflicting, about an equal number of workers reporting for and against the value of spraying. At present it seems, however, to be the general opinion that spraying is of little value. In preventing the disease, or in reducing it to the lowest amount, the following points should serve as a guide:

1. Some relief may be obtained by the selection of varieties that show more or less resistance.
2. Overvigorous plants show more susceptibility to the disease than plants which are making a slower and less luxuriant development.
3. A continuous uniform growth is more likely to give freedom from the disease than fluctuating or intermittent growth.

4. Shortage of moisture at the critical time in the development of the fruit is one of the most important causal factors.[1]

5. Excessive watering may greatly increase the disease over that shown by plants which have received a moderate supply.

6. Heavy applications of barnyard manure have increased the disease, especially in certain soils.

7. The relation of artificial fertilizers to the disease is somewhat variable, and positive recommendations are unsafe.

8. The disease is increased by raising the soil temperature in greenhouse culture.

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IMPORTANT DISEASES DUE TO UNFAVORABLE WATER RELATIONS

Bitter pit.—(See special treatment, pp. 102–114.)

Black end of pears.—This disease seems to be connected with the use of Japanese rootstocks which interfere with the movement of water from the rootstock to the scion. It appears to be aggravated by high temperatures and low humidity, with the resultant heavy demand on water. HEPPNER, M. J.: Pear black end and its relation to different rootstocks. *Proc. Amer. Soc. Hort. Sci.* **24**: 139–142. 1928. TUFTS, W. P. AND DAVIS, L. D.: Hard end or black end of pears in California. *Proc. Wash. State Hort. Assoc.* **25**: 108–115. 1930.

Blossom-end rot of tomato.—(See special treatment, pp. 114–121.)

[1] Valleau and Johnson (*Kent. Res. Bul.* **281,** 1927) have suggested that drought spot, cork and bitter pit of apples and blossom-end rot of tomatoes are due to a nitrogen starvation of the affected tissues.

Cork of apples.—Affected fruits show internal brown, dry, spongy or corky patches which may or may not be evident by external irregularities (see Fig. 43). The variability of the symptomology and the study of these troubles in widely separated localities have led to the use of such names as "malformation," "dry rot," "York spot," "punky disease," "hollow apple," "crinkle" and confluent bitter pit. MIX, A. J.: Cork, drought spot and related diseases of the apple. *N. Y. (Geneva) Agr. Exp. Sta. Bul.* **426**: 473–522. 1916. See also BROOKS and FISHER, and FISHER under Drought Spot.

Drought spot or spot necrosis of apples and prunes.—Drought spot of apples may be caused by a sudden and pronounced shortage of water following a normal supply earlier in the season, resulting in the formation of large, irregular, somewhat depressed, water-soaked lesions, generally near the calyx end. In prunes, it appears first as a watery spot beneath the skin, followed by degeneration of the affected tissue with the formation of gum which may burst the skin and ooze to the surface. BROOKS, CHARLES AND FISHER, D. F.: Irrigation experiments

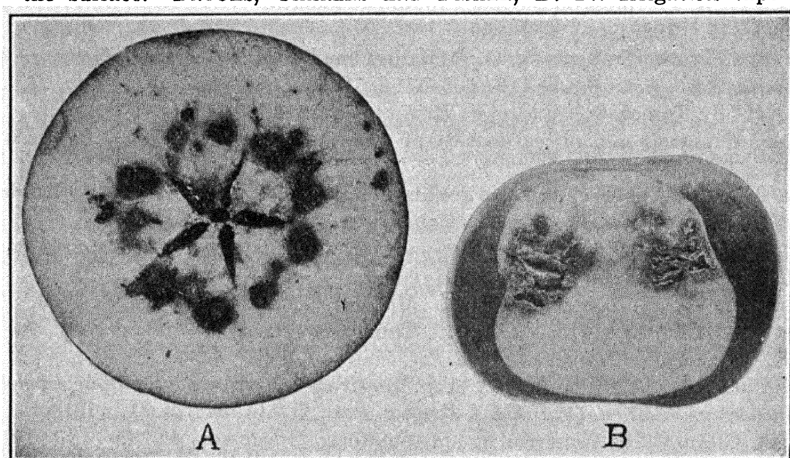

FIG. 43.—*A*, cross-section of King apple affected with cork, showing brown corky tissue near the core; *B*, section of a York Imperial, showing pockets and brown corky tissue beneath the surface depression. (*After Brooks and Fisher.*)

on apple-spot diseases. *Jour. Agr. Res.* **12**: 130–131. 1918. FISHER, D. F.: Drought spot and related physiological diseases. *Proc. Wash. State Hort. Assoc.* **16**: 35–39. 1920.

Hollow heart of potatoes.—Characterized by an internal split or cavity resulting from rapid growth induced by an abundance of moisture and food materials. MOORE, H. C.: Further studies of potato hollow heart. *Mich. Agr. Exp. Sta. Quart. Bul.* **11**: 20–24. 1928.

Internal decline of lemons (*Endoxerosis*).—Characterized by an internal necrosis and destruction of tissues usually at the stylar end, frequently beginning in the *green* stage or the *silver* stage but becoming especially marked in the *tree-ripe* or *yellow* stage. BARTHOLOMEW, E. T., BARRETT, J. T. AND FAWCETT, H. S.: Internal decline of lemons. I. *Amer. Jour. Bot.* **10**: 67–70. 1923. ——: Internal decline of lemons. II. *Amer. Jour. Bot.* **13**: 117–126. 1923. III. *Ibid.* **13**: 102–117. 1926. —— AND ROBBINS, W. J. IV. *Ibid.* **13**: 342–354. 1926. V. *Ibid.* **15**: 497–508. 1928. VI. *Ibid.* **15**: 548–563. 1928. FAWCETT, H. S.: Endoxerosis or internal decline of the lemon. *In* Citrus Diseases and Their Control, pp. 421–426. McGraw-Hill Book Company, Inc., New York. 1926.

Straight head of rice.—The heads develop slowly, remain green longer than normal and stand erect when normal heads are drooping. The glumes or other parts of the inflorescence are aborted, distorted or perfect, but complete flowers still remain sterile. The disease can be prevented by proper control of the water. TISDALE, W. H. AND JENKINS, J. W.: Straight head of rice and its control. *U. S. Dept. Agr. Farmers' Bul.* **1212**: 1–16. 1921.

Water core of apples.—This trouble is characterized by the presence of glassy or watery areas in the flesh or pulp located near the core or extending out toward the surface. The glassiness results from the filling of the intercellular spaces with cell sap. O'GARA, P. J.: Studies on the water core of apple. *Phytopath.* **3**: 121–128. 1913. FISHER, D. F.: Water core. *Proc. Wash. State Hort. Assoc.* **19**: 98–104. 1923. BROOKS, C. AND FISHER, D. F.: Water core of apples. *Jour. Agr. Res.* **32**: 223–260. 1926. FISHER, D. F., HARLEY, C. P. AND BROOKS, C.: The influence of temperature on the development of water core. *Northwest. Fruit Grower* **3**: 8, 11. 1931.

White spot of alfalfa.—Two types of lesions characterize the disease: (1) localized spotting and (2) marginal injury. Both types may be so severe as to involve the entire leaf surface. An unbalanced water relation induced by irrigation is thought to be the principal cause, but heavy rainfall following drought may be a contributing factor. RICHARDS, B. L.: White spot of alfalfa and its relation to irrigation. *Phytopath.* **19**: 125–141. 1929.

CHAPTER VI

DISEASES DUE TO IMPROPER AIR RELATIONS

Living plants or plant structures stand in intimate relation to their air environment from which certain materials must be obtained and to which by-products of their activities are contributed. Unfavorable air relations may interfere with income and outgo of gaseous materials and thus induce disease.

General Air Relations of Plants and Plant Structures.—Every living portion of a plant body must be so related to its environment or to other plant parts as to obtain a supply of oxygen. In other words, every living part of the plant breathes. In this process of respiration, complex chemical changes are taking place within the living cells, but certain end products mark the process, carbon dioxide being given off and oxygen consumed. Oxygen is taken in through aerial parts and diffuses throughout an intercellular system of connecting spaces; it is also absorbed by the root system of the plant from the air in the spaces between soil particles. In the case of our common crop plants the roots cannot obtain their needed oxygen from or through the aerial parts but must obtain it direct from their soil environment. Respiration increases in the seed with absorption of water and exposure to favorable temperatures and continues as an active process throughout the life of the plant. In dormant plants or plant structures like tubers, rhizomes or fleshy fruits, etc., in which the life processes are at a low ebb, respiration does not cease until life is extinct. Any pronounced interference with the respiratory exchanges will result in lowered efficiency and, if continued, in evident ill health and final death.

It is only under exceptional circumstances that the breathing of the aerial parts of our growing crops is likely to be seriously interfered with. A heavy deposit of dust or of inert particles like cement dust may coat the aerial parts and choke the stomata of leaves and thus cause pronounced injury by interfering with the exchanges of gases. Dormant structures like tubers or bulbs or languid organs like fruits, when removed from their natural environment and collected in closely crowded quarters of the storehouse or the market package, may suffer from the lack of sufficient aeration. The oxygen requirement may not be satisfied, or the stagnation of the air may delay the removal of volatile products of protoplasmic activity.

Since underground parts must get their oxygen from the soil air, and since there are many ways in which the air content of the soil may be

lessened or its free circulation impeded, asphyxiation of roots is a fairly common phenomenon. This may cause pronounced disturbances in the life of the plant or may end in its death. Most cultivated plants cannot obtain sufficient oxygen from water; hence a soil saturated with water will not supply sufficient oxygen to the root system. This will explain in the main the injurious effects of heavy, poorly drained or water-logged soils, "wet feet," flooded lands or other conditions in which water drives out the soil air which is so essential to a healthy development. The physical structure of the soil may be such as to impede the circulation of the air so that the oxygen supply may be used faster than it is replaced, or the position of the root system may be such that new supplies of oxygen do not reach them rapidly enough. This will explain the injurious effects of closely compacted or hard soils, silt deposits, surface coverings of cement walks or pavements, deep seeding or setting of plants or filling around trees in grading for the construction of buildings. Seeds may fail to germinate, growing annual crops may sicken and die or lead a struggling life or trees may blight or die back because of the inability of the root system to obtain the necessary air.

APPLE SCALD

This storage and transportation disease of apples is characterized by certain skin discolorations followed by internal changes which disfigure the fruit and shorten the duration of its storage period.

History and Geographic Distribution.—Scald of apples is a trouble that must have occurred ever since apples have been held in storage, but it is only in recent years that it has been recognized by growers, shippers and plant-disease specialists as a disease of great commercial importance. While it has occurred in common storage, it has been much more in evidence since the extensive refrigeration of fruit in transit to market or during storage. The disease was of sufficient importance in Vermont in 1896 (Jones) to demand consideration, but it had previously been known as scald, and its prevalence recognized, especially on certain varieties. The disease was well characterized, and its cause discussed in the above-mentioned report and also a year later (Jones and Orton, 1897–1898), but little advance was made in the knowledge of its etiology. It was recognized as a trouble of non-parasitic character, and it is interesting to note one of the conclusions that "it appears certain that the primary cause of the scald must be sought in the climatic and orchard conditions, the conditions of the storehouse being secondary." Time has shown the truth of the statement that "apple scald presents a complex problem, the solution of which needs the accumulated observations and experiments of a series of seasons."

Scald was given special attention in the studies by the Office of Pomological Investigations of the U. S. Department of Agriculture on the general problem of "the apple in cold storage" (Powell and Fulton, 1903) and also received consideration in New York (Beach and Clark, 1904) and later in Iowa (Beach and Eustace, 1909; Greene, 1913; Whitehouse, 1919; Plagge and Maney, 1924, 1925). The most detailed studies on scald have been made by the plant pathologists of the U. S. Department of Agriculture (Brooks, Cooley and Fisher, 1917 and later), and we are indebted to them for the establishment of the real cause of the trouble and for the perfection of the oiled wrapper as a means of control. The Pacific Northwest, as a large producer

of apples, has been the center for some of these recent studies (Ramsey *et al.*, 1917; Fisher, 1920).

Symptoms and Effects.—Scald is characterized by the discoloration of the skin of stored fruit, the color varying from a faint-brown tint in mild cases to a pronounced brown involving the entire thickness of the skin in more severe cases. The scald appears first on the lighter surface of the fruit, where it is most severe and spreads until all or a large part of the surface may be involved, depending somewhat upon the maturity of

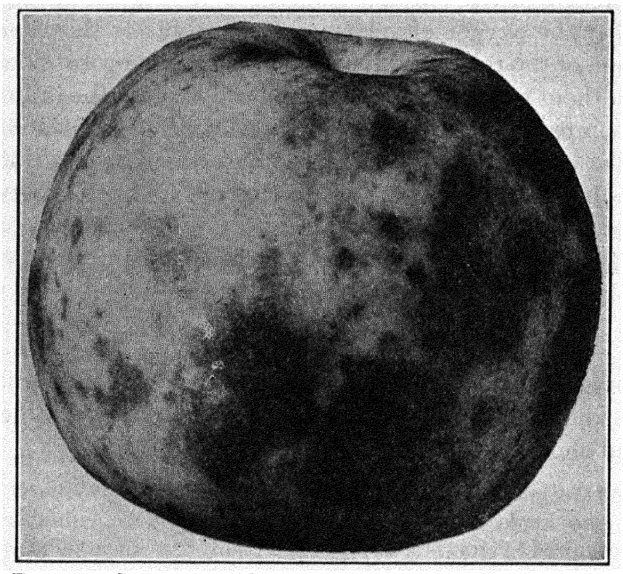

FIG. 44.—Common scald of apple. (*After Brooks and Fisher.*)

the fruit at the time of storage. Green or undercolored skin is the most susceptible, yellow of medium susceptibility, while deep red is generally the most resistant.

In the very severe cases, the entire skin layer is killed and sometimes broken down to the extent that it will slough off readily from the flesh. In some instances, the flesh becomes dead and brown to a depth of ½ inch and the disease takes on the appearance of an apple rot; but true rot usually spreads down into the flesh in a more or less conical shape, while scald is usually diffuse, spreading over a large area without having much depth (Brooks, Cooley and Fisher, 1920).

Severe scald may be followed by physiological decay or internal breakdown, which may involve much of the interior. The scalded areas are soon invaded by rot-producing fungi which find the dead tissues an easy avenue of entrance and thus complete the work of destruction.

The symptoms described above have sometimes been designated as *common*, *superficial* or *hard scald* to distinguish them from another trouble of somewhat different origin known as *soft* or *deep scald* (see p. 138).

In both common scald and soft scald, the injured tissues may be invaded by Cladosporium or other fungi and become spotted with black.

While scald may appear in common or home-stored fruit, it is of most concern as a disease of commercial storage or of market fruit.

Scald may appear on apples while still in commercial storage, but it is only after they have been moved to the warmer temperature of the market or the home that it makes its most rapid development. Apples may appear to be in perfect condition upon removal from storage and yet a few days later have their market value reduced 15 to 30 per cent or more on account of the development of scald. A disease that makes such a sudden appearance at a time when the apples

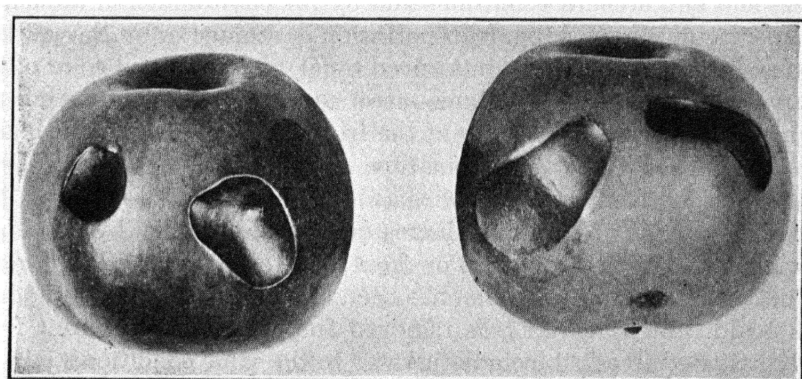

FIG. 45.—Soft scald of Jonathan apples. (*After Whitehouse, Iowa Exp. Sta. Bul.* **192.**)

are ready for consumption naturally has a very disturbing effect upon market operations, resulting in heavy losses and tending to limit distribution and decrease consumption (Brooks, Cooley and Fisher, 1923).

It has been stated that market inspection reports show that scald causes almost as much loss on the market as blue mold. This does not take into account latent scald that appears later; hence the total losses are probably higher than from any other market trouble. Apple scald causes losses or handicaps to the apple industry in a number of ways: (1) by depreciation in price due to actual occurrence of the disease or to anticipation of loss from its increase; (2) by spoilage before the fruit can reach the consumer; and (3) by restricting purchases when scald is likely to develop and thus affecting market conditions and lessening total consumption.

Etiology.—Apple scald is a non-parasitic or physiological disturbance due to the production, by the tissues of ripening or aging fruit, of volatile esters which have a toxic effect upon the tissues if conditions are such that they accumulate in the tissues or in the surrounding air. This conclusion is based on the fact that typical scald can be "artificially produced in a few days' time by exposing apples to the vapors of ethyl acetate, amyl acetate or methyl butyrate" and on the protecting effect of circulating

air or of substances which can absorb these volatile products and so prevent their accumulation. It was suggested at one time that the scalding might be due to a shortage of oxygen or to an excess of carbon dioxide, because of the massing of large quantities of fruit during storage, but it has been shown that scald will develop when there is no shortage of oxygen and will not be produced by an excess of carbon dioxide. On the contrary, it has been found that "high percentages of carbon dioxide delay the ripening of the apples and greatly decrease the development of scald" (Brooks, Cooley and Fisher, 1923).

The time of appearance and the severity of the trouble are influenced by seasonal and orchard conditions and by the environmental influences that operate during packing, transportation or storage or on the market. The susceptibility to scald is influenced by (1) maturity and color of the fruit at picking time; (2) the amount of moisture available during the growing period; and (3) the size of the fruit. Well-matured and highly colored fruits scald less than immature or poorly colored fruits. Fruit that is picked green may show twice as much scald as well-matured, but not overripe, fruit. Apples are increased in susceptibility by abundant moisture either from irrigation or from natural rainfall. The forcing that may follow heavy irrigation has been known to cause three times as much scald after the fruit was removed from storage as in fruit from lightly irrigated trees subjected otherwise to the same conditions. Large apples are generally more susceptible to scald than small ones, mainly because of a forced growth, immaturity and poorer color.

The development of scald is influenced by (1) the temperature to which the fruit is subjected after picking, during storage or during transit to market; (2) the aeration or ventilation of the fruit; and (3) the humidity of the air to which it is exposed.

The harvested apple is still carrying on its physiological processes and will continue to do so as long as its tissues are living. It must be evident that these processes, which are essentially chemical reactions, will be speeded up by high temperatures or slowed down by low temperatures. Since scald is a chemical phenomenon, it is but natural to expect that temperature conditions which retard respiration and tissue activity in general would delay the appearance of the disease. Storage at low temperature does not prevent scald, but it delays its onset, simply by slowing down the life processes. The influence of temperature upon scald may be illustrated by the results obtained by numerous investigators that immediate storage at 32°F. resulted in more delay in the appearance of the disease than storage at any higher temperatures, especially if the fruit was in prime condition at picking. With fruit that has reached prime maturity at picking time, the appearance of scald is advanced by delayed storage or by holding at temperatures higher than the storage temperature. Immature fruit will scald less by holding at "ordinary

temperatures until it more nearly reaches the best or optimum degree of maturity for cold storing" (Whitehouse, 1919).

Stagnation of the air is conducive to the development of scald; hence piling apples in large heaps, close ranking of containers, use of tight containers or storage in poorly ventilated warehouses or storage rooms will favor the onset of the disease. If delayed storage is necessary, it is important to provide conditions which will give the best possible circulation of the air, as by this means the incidence of the disease will be delayed when the fruit goes into cold storage. The importance of aeration in the storage room is emphasized by the fact that scald is often less in well-ventilated cellars and air-cooled storage rooms than in commercial cold storage. The circulation of the air is the important feature rather than the introduction of fresh air. It has been noted that apples near the aisles or doors of a storage room are less affected than those in the center of the stacks. Crowding in a poorly ventilated room will give a large amount of scald, but if, in addition, tight containers are used, scald will be still further increased. It has been shown that apples in ventilated barrels, baskets or hampers scald much less than fruit in tight barrels, the difference being due in part to the more rapid cooling in the ventilated packages but mainly to the free access of air. For this same reason, boxed apples scald less than barreled stock, unless the boxes are stacked too tightly to permit a circulation of the air. The early part of the storage period, *i.e.*, the first 6 or 8 weeks, is an especially critical time in the storage life of the apple, and during this period good aeration and ventilation are essential. It has been shown that scald is increased by high humidity of the storage room, but the ventilation and the temperature have more influence. The maximum amount of scald will develop in immature fruit from heavily irrigated orchards, packed in unventilated containers and held at high temperatures in humid, poorly ventilated storage rooms.

Susceptibility of Varieties.—Under unfavorable conditions, scald may occur on almost any variety of apple, but certain varieties are especially susceptible. It is generally agreed that green and yellow are more susceptible than red varieties, although in some red varieties the disease becomes of commercial importance. Partially colored fruits scald mainly on their green or yellow surface. Beach and Clark (1904) report Grimes, Mann, Tolman Sweet, Winter Banana, Yellow Bellflower, Rhode Island Greening and Green Newtown as the most susceptible yellow varieties and Baldwin, Gano, Missouri Pippin, York Imperial and Winesap as the most susceptible red varieties, as a result of their storage studies in New York. Whitehouse (1919) lists Grimes, Mammoth Black Twig, Sheriff, Northwestern Greening, Willow Twig and less commonly Winesap as the principal varieties on which scald is commercially important in Iowa. Other varieties which have been listed as especially

susceptible are the Rome Beauty, Stayman Winesap, Wagener and Baldwin.

Prevention.—While the scald of apples cannot be absolutely prevented, it can be held in check to such an extent that market losses can be nearly eliminated. While some few practices have proved of outstanding value, the progressive fruit man will give attention to both preharvest and after-harvest factors. The following control features should be kept in mind and followed as closely as possible to secure the production and marketing of a high-grade product: (1) Allow fruit to reach prime maturity before picking, and follow cultural practices to produce the best color possible for the variety. Avoid, especially, undermatured or overstimulated stock except for early consumption. (2) Avoid delayed storage of apples that have reached prime maturity, but if delay is necessary provide for free access of air during the prestorage period, and protect as much as possible from the direct sun or high temperatures. Hold immature fruit until it approximates prime maturity before putting into cold storage. (3) Store fruit as quickly as possible after picking, and reduce the temperature promptly. As nearly as possible, maintain the temperature of the *stored fruit* at 32°F. and the relative humidity of the air in the storage room between 80 and 85 per cent. In some localities, certain varieties like the Yellow Newtown, for example, may require a slightly higher temperature (36 to 40°) to prevent "internal browning."[1] (4) Arrange for as thorough and complete aeration of the storage room as possible, keeping in mind that the movement of the air is more important than the introduction of fresh air. (5) Avoid the use of tight containers, and provide open stacks in the storage room. The use of ventilated or open containers helps to retard the appearance of the disease. (6) Practice wrapping of the fruit, using special oiled paper wrappers. Experimental tests have shown that ordinary paper wrappers greatly reduce apple rots in storage (Powell and Fulton, 1903) and are of some value in the prevention of scald (Whitehouse, 1919), but almost complete control is obtained with the use of the oiled wrappers as reported by Brooks, Cooley and Fisher (1923).

The control of scald by means of oiled wrappers might seem to be in contradiction to the principles of scald control developed under the subject of aeration. It was pointed out, however, in the earlier discussion, that high percentages of carbon dioxide tend to delay the ripening of the apple and to decrease scald, while high percentages of the odorous gases thrown off by the apple are injurious to the skin of the apple. The oil removes these odorous substances by absorption, in the same manner that butter and other fats take up various odors, and the oiled wrappers as they come from the package are heavily charged with the various odorous materials thrown off by the apples. The oil also has a checking effect upon the life activities of the skin of the apple, slightly delaying the development

[1] See *U. S. Dept. Agr. Bul.* **1104,** 1922; also *Cal. Bul.* **370,** 1923.

of yellow in the ground color and probably at the same time checking the development of scald.

Two other methods of using the oil have been tried: first, coating the skin of the apple (Brogdex treatment), and, second, scattering shredded oiled paper between the apples. The first method has sometimes injured the appearance and flavor of the fruit, but since the perfection of the Brogdexing machines this defect has been largely overcome. The second, although less efficient than the oiled wrappers, has been found well adapted for the control of scald on apples packed in barrels, hampers or other containers of the unwrapped fruit.

The most important of the control practices are (1) the storage at low temperatures; (2) the use of oiled paper as wrappers or in shredded strips scattered between unwrapped apples; and (3) coating the skin of the apples with oil by the Brogdexing process. By these practices, scald is reduced to a minimum.

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BLACKHEART OF THE POTATO

The potato is affected by a number of diseases marked by a discoloration of the interior tissues of the tuber, but one of these internal necroses which causes a characteristic blackening of the center and sometimes of more external parts has been called the blackheart or, less frequently, heart rot. Internal discolorations of the tuber may be caused by either parasitic or non-parasitic influences.

General Types of Internal Tuber Troubles.—The principal groups of diseases which cause internal necrotic areas and their important characters will be briefly presented:

1. Decays due to the action of either bacteria or fungi, including blackleg tuber rot, slimy soft rot, fungous rots caused by various species of Fusarium, jelly-end rot, a soft watery rot called leak or melters, late-blight rot and some others of minor consequence.

2. Vascular discolorations frequently followed by decay, including bacterial wilt or brown rot of the southern states, Fusarium blight caused by various Fusarium species and Verticillium wilt. In all three troubles, the initial tuber lesions are in the nature of brown discolorations of the vascular ring especially at the stem end, while a later phase in the nature of a rot may be induced by the advance of the causal organism into tissues adjacent to the vascular ring.

3. Minute discolored areas below the skin which accompany a knotty or irregular surface, characteristic of the attacks of the root-knot nematode, or eelworm (see Nematode Diseases, Chap. XXVIII).

4. Phloëm necrosis in the nature of a network of brown strands extending throughout the flesh of the tuber, especially of the stem end, one of the tuber symptoms of leaf roll, a virus disease (see Chap. XII).

5. Non-parasitic troubles, including simple stem-end browning of the vascular ring; internal brown spot characterized by brown spots scattered throughout the flesh of the tuber (identical with "sprain" of English writers and "Buntwerden" or "Eisenfleckigkeit" of the Germans); heat and drought necrosis in the nature of a yellowing and browning of the vascular ring and the more external tissues; sunburn or greening due to exposure to light; sun scald, or the killing of external tissues from expo-

sure to intense heat and light; freezing injury and frost necrosis, evident as ring, blotch or net types of discoloration; hollow heart, or a central cavity lined by dead-brown cells; and blackheart, the subject of this section. It should be understood that a given lot of tubers may show more than a single internal disturbance and that non-parasitic lesions like hollow heart, frost injury, sun scald and blackheart are frequently invaded by bacteria or fungi which find the dead or injured tissues a very congenial substratum.

History.—While the blackheart of potatoes has undoubtedly been a cause of loss for many years, it was not recognized as a specific trouble to which the name blackheart was applied until the work of Bartholomew (1913, 1915). This trouble

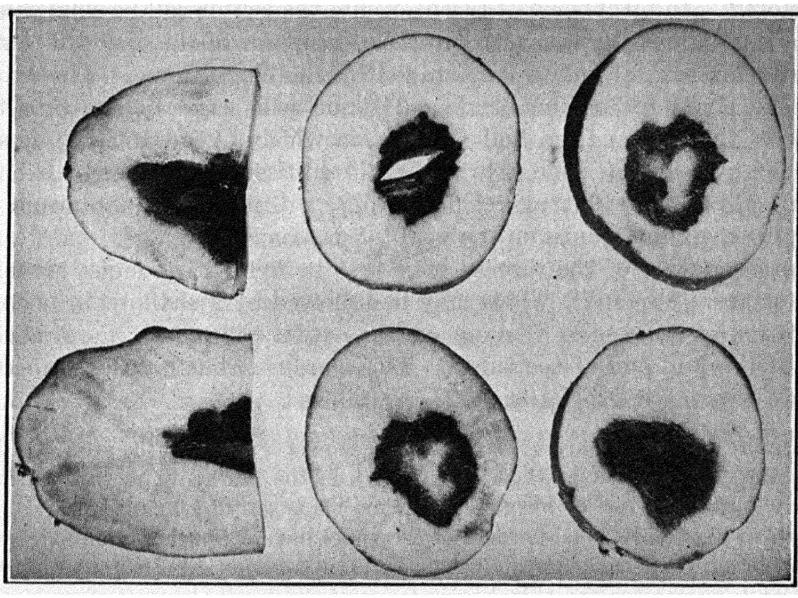

FIG. 46.—Blackheart of potato.

was first brought to the attention of plant pathologists by its appearance in severe form in carload shipments occurring either while in transit or soon after reaching their destination. The experience of shippers that the trouble was due to overheating in transit was confirmed by the studies of Bartholomew, and this was accepted as the sole cause until the publication of the bulletin by Stewart and Mix (1917) on "Blackheart and the Aeration of Potatoes in Storage," in which the disease was shown to be of wide occurrence and the result of a deranged or deficient respiration influenced by temperature and the available supply of oxygen. More recently, a chemical study of the blackheart of the potato has been made by Mann and Joshi (1921), and Coons (1924) has given special attention to the injurious effects of the disease upon seed stock.

Symptoms and Effects.—The appearance of tubers affected by blackheart is exceedingly variable, depending upon the exact conditions under which the disease has developed. In one form of the disease, the tubers may appear perfectly normal as far as external characters are concerned,

but when cut in two they are found to show a browning or blackening of the interior. The discoloration generally starts at the center and progresses toward the outside, causing either a star-like radiation or a more uniform advance. If the inciting conditions operate for a sufficiently long period, the blackening may advance until it reaches the surface. The blackened tissues are in sharp contrast to the normal flesh, similar in consistency to normal tissues or more firm or slightly leathery if they have partially dried. This character should distinguish the lesions from those of either leak or black rot, which are similar in color but soft and watery. In some cases of blackheart, the discoloration appears in the form of zones surrounding the center, which is normal or only slightly discolored. In blackheart of recent origin, the center will be solid; but if of some standing (10 days or more), the shrinkage of the diseased tissues will cause a central hollow surrounded by the black tissue. The extent and the character of the blackened tissue will serve to separate this phase of the disease from hollow heart, in which a growth cavity is surrounded by a narrow zone of brown, oxidized tissue. If these blackheart lesions advance until they reach wounds, rot-producing organisms are likely to enter and continue the work of destruction.

Another type of the disease may first be evident as moist areas on the surface ("sweat"). This may be followed by a shallow, brown discoloration over areas of varying surface extent, frequently more noticeable at the bud end of the tuber. This condition has been described as *surface breakdown* by Kotila (1923) as follows:

> The first symptoms are observed by the grower 3 or 4 months after the date the potatoes were stored, when some tubers in the bin show on their surfaces slightly sunken, round or irregularly shaped spots varying in size from 1/16 to 3/4 inch in diameter. The borders of the spots have a bluish or gunmetal hue. Upon cutting these sunken spots with a knife, it is found that they are only skin deep, and, except for the thin brown layer of dead cells beneath the skin, no rotting has progressed into the tuber. This surface breakdown is the so-called "button rot" of the trade, but it is not a true rot of the tuber. The pitting of the tubers becomes more pronounced as the season advances and, with the coming of warm weather in the spring, the most severe symptoms of breakdown are seen.

If conditions continue favorable to the disease, internal discolorations of typical blackheart will also appear. If affected tubers are cut before the inciting cause has been acting too long, the flesh will be nearly normal, but later it turns pink on exposure to the air and then gray black or purplish to coal black. Under unfavorable conditions, even fairly early in the season, many of the affected tubers will be invaded by either bacteria or fungi, and heavy losses may result.

The potatoes affected by blackheart, which are not invaded by rot-producing organisms and destroyed, are sometimes ruined for use as table stock owing to the extent of the internal discolorations and are also

rendered unfit for seed purposes. According to Stewart and Mix (1917), potatoes severely affected with blackheart may produce apparently normal sprouts, and these investigators state that such tubers are unfit for seed but that slightly affected tubers may be planted. Coons (1924) emphasizes the undesirability of affected tubers for seed purposes and presents evidence that they may be the cause of the ragged appearance and uneven stand in many potato fields. He attributed this behavior to the "bacterial decay that destroys the seed pieces before the young plant has become established in the earth." Later studies have shown that germination, vigor and yield are inversely proportional to the degree of blackheart.

Etiology.—The first studies of blackheart showed that the blackened tissues of affected tubers were free from any organisms, and therefore the disease was believed to be the result of a derangement of normal physiological processes due to certain environmental factors. It has been shown that blackheart is really due to an asphyxiation of the tissues of the tuber due to lack of sufficient oxygen. This lack of oxygen is much more likely to be effective in causing the trouble at high temperatures when the life processes are speeded up than at more moderate temperatures, but under these less favorable conditions the disease may develop if the tubers are not sufficiently aerated. It was first shown by Bartholomew (1913–1915) that the disease could be artificially produced by subjecting tubers to a temperature of from 38 to 48°C. for 14 to 48 hours, 42 to 44°C. being the optimum. It is significant that this optimum temperature is very close to the temperature range for maximum respiration. This treatment produced the typical internal discolorations that have been the cause of loss in numerous carload shipments. Even before this experimental proof, it was the belief of shippers that the trouble was due to overheating in transit. Potatoes shipped during cold weather must be protected from freezing injury, so it has been the custom to use either refrigerator cars heated by wood stoves or special cars with automatic heat control. With the stove method of heating, the regulation of the temperature is impossible or very difficult, and cases of overheating are not uncommon. In 1914, the writer inspected a car of potatoes at Atlantic City, N. J., that had been shipped from Maine in an Eastman heater car and found that the entire carload was ruined by blackheart. The automatic temperature control had failed to function, and consequently the car was overheated during transit. There are also plenty of cases on record in which the disease has appeared as the result of overheating potatoes held in common storage.

It was accidentally discovered by Stewart and Mix (1917) that "by excluding the air from potatoes, blackheart may be produced at temperatures much lower than those employed by Bartholomew." This led to a more detailed experimental study of the air requirements of the potato

during its storage period and emphasized the fact that a "considerable volume of air is required for the well-being of potatoes." The first work with tubers in sealed jars showed that "within a certain length of time, which varies with the temperature and quantity of air available, tubers confined in hermetically sealed jars" develop the first symptoms of surface breakdown and with later exposure to the air the external discolorations and the internal discolorations of typical blackheart. The length of time required to produce the symptoms was increased with the exposure to lower temperatures, but the blackheart developed even at 40°F. after 23 to 40 days when the volume of air was equal to the volume of the potatoes. It was also shown that tubers may suffer from insufficient aeration due to deep piling and that under such adverse conditions they may behave essentially like tubers in sealed jars. "They sprout feebly or not at all, become moist on the surface, discolor externally upon exposure to the air and are often affected with blackheart internally." It has been claimed that the injury resulting from poor aeration is due to a lack of oxygen and not to the accumulation of carbon dioxide given off in respiration, but blackheart has been produced artificially in CO_2-free air with an abundance of oxygen (Davis, 1926). From these studies and general observations, it is certain that blackheart is found quite frequently in common storage. Heaviest losses have been reported during seasons of bumper crops, with the crowding of poorly ventilated houses, even though the temperature was held at 40° or below.

According to Bartholomew (1915), the heated or asphyxiated tissues of the tuber develop an increased amount of the aromatic amino acid *tyrosin*. The enzyme *tyrosinase* is present also, and the interaction of the two results in the formation of a black precipitate, which has been called "melanin" or "humin." The blackening of the tissues is due, then, to (1) the increase in the amount of the chromogen tyrosin in free form; (2) the access of an unusual amount of oxygen, due to the killing of the cells; and (3) the accelerated action of the oxydizing enzyme *tyrosinase*. As a result, the affected tissues undergo a series of color changes ranging from light pink to coal black. According to Davis (1926), there is an accumulation of CO_2 and a depletion of oxygen in the tissues previous to the appearance of the discoloration. There is a high respiratory activity, and exchange of gases fails to keep pace with the respiratory rate. It is also stated that temperatures above 38°C. may have a direct effect, since this is the critical point for the maintenance of normal water relations.

Prevention.—The recognition of blackheart as due (1) to exposure to high temperatures during storage or transit to market or (2) to crowding in poorly aerated storage rooms should at once suggest the methods of prevention. It should be emphasized that temperature control alone during the storage period with no attention to aeration is not sufficient.

Probably first attention should be given to storing the potatoes in such a way that they will be well supplied with air, and it should be remembered that the higher the temperature the more the need of a good circulation of air. Storage in deep bins or in continuous high ranks of sacks is likely to induce blackheart. As an insurance against its development, the potatoes to be held for 6 months or more should not be piled more than 6 feet deep even when the storage temperature is held around 45°F., or, in other words, no potatoes should be more than 6 feet from an open-air space. If the storage temperature is likely to range from 50 to 70°F. for longer than 3 weeks during the storage period, no potatoes should be more than 3 feet from an open-air space. To increase the storage capacity of a given floor space, false floors and walls to the bins, with spaces between, may be provided. In sack storage, spaces between ranks of sacks will accomplish the same results. With these arrangements and with inlets for fresh and outlets for foul air, blackheart should be prevented unless the potatoes are exposed to abnormal temperatures from artificial heating.

Even with the best provision for ventilation, the temperature of the car or of the storage room should not be allowed to go over 95°F., and in heated cars it will be best to hold it at 60°F. or lower. For further data on the minimum temperatures permissible, see Frost Necrosis (p. 172).

Potatoes should not be left long in soil after the vines are dead, if in a region of high soil temperatures; neither should they be left lying long in the hot sun after digging. Care should also be taken not to hold freshly cut potatoes in large piles, as blackheart may develop under such conditions.

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Important Diseases Due to Improper Air Relations

Blackheart of potatoes.—(See special treatment, pp. 132–137.)

Black leaf speck of crucifers.—This is a disease of cabbage, cauliflower and other crucifers which appears during storage and transportation to market. It is characterized by small lead-grey to black specks. NELSON, R.: Storage and transportational diseases due to suboxidation. *Mich. Agr. Exp. Sta. Tech. Bul.* **81**: 1–38. 1927.

Button rot of potato.—Shallow surface pitting caused by death and desiccation of areas of surface tissue; frequently followed by Fusarium decay. NELSON, R.: *Loc. cit.*

Red heart of lettuce and cabbage.—Heart leaves of lettuce develop a deep chestnut-brown color and cabbage a typical red color. NELSON, R.: *Loc. cit.*

Internal browning of the Yellow Newton apple.—This defect appears in cold storage as brown streaks in the pulp radiating outward from the center. It is closely related to scald. OVERHOLSER, E. L., WINKLER, A. J. AND JACOB, H. E.: Factors influencing the development of internal browning in the Yellow Newton apple. *Cal. Agr. Exp. Sta. Bul.* **370**: 1–40. 1923.

Brown heart of apples and pears.—This disease has caused heavy losses in Australian shipments to England and is attributed to the excess of carbon dioxide in the hold of the vessels, accompanied by a low oxygen concentration. Experiments have shown that "the danger limit of carbon dioxide is lower when the temperature is low; thus a concentration of carbon dioxide which will have no effect at higher temperatures will cause brown heart at lower temperatures." KIDD, F. AND WEST, C.: Brown heart—a functional disease of apples and pears. *Dept. Scientific Ind. Res. Food Investigation Bd. (London). Special Rept.* **12**: 1–54. 1923.

Apple scald.—(See special treatment, pp. 125–132.)

Soft scald of apples.—Soft scald varies from small spots ⅛ inch or less across to large areas that sometimes include nearly the whole surface of the apple. It may be compared to "the effects produced by touching or rolling an apple on a hot stove—the sharply defined margins, tightly drawn skin and 'cooked' appearance of the affected tissue are all very similar" (Fisher). The scalded areas frequently form more or less elongated transverse lesions or peculiar irregular patterns. A trouble designated by Whitehouse (1919) as "dry brown rot" is apparently soft scald (see Fig. 45). Jonathan, Blue Permain and Wealthy are particularly susceptible. Soft scald is not controlled by oiled wraps but has been greatly reduced by the Brogdex treatment (Heald, 1931) and by immediate packing and cold storage (Harley and Fisher, 1931). The production of soft scald by delayed packing following harvesting has not been substantiated by recent tests by the author. FISHER, D. F.: (See Scald.) WHITEHOUSE, W. E.: (See Scald.) HARLEY, C. P. AND FISHER, D. F.: A study of the internal atmosphere of the apple in relation to soft scald. *Proc. Am. Soc. Hort. Sci.* **27**: 271–275. 1930. HEALD, F. D.: The control of soft scald. *Better Fruit* **25**: 12. 1931.

CHAPTER VII

DISEASES DUE TO HIGH TEMPERATURES

The life of a plant is a complex of physiological processes which are operating according to chemical and physical principles: absorption, food manufacture, digestion of foods, assimilation, translocation of food, respiration, transpiration and minor life phenomena which lead to growth, maintenance of life and reproduction. All of these life processes are influenced by the temperature to which the plant is exposed, and this may be illustrated by the relation of temperature to growth. Life is possible only within the limits of certain temperatures, and growth has an even narrower temperature range.

General Temperature Relations of Plants.—There are three *cardinal points* in the temperature relations of any species of plant:

1. A *minimum* temperature or degree of warmth, at which growth first begins.
2. An *optimum* temperature, at which the growth is most rapid.
3. A *maximum* temperature, beyond which growth ceases.

As the temperature rises from the minimum, the growth is gradually increased up to the optimum, while beyond the optimum the growth becomes slower and slower until the maximum is reached and it ceases. Life may continue below the minimum, the plasma body passing into a *cold rigor*, but death may result if the temperature drops too low, *i.e.*, below the *subminimum;* or, again, the plant may survive above the maximum growth temperature, the living substance existing in a state of *heat rigor*, but succumb if the temperature becomes too high or is maintained for too long a period above the supramaximum. Since every physiological process has its own cardinal temperature points, it must at once be apparent that thriftiness of growth and productiveness of a crop are influenced by the temperatures to which it is exposed. The various organs of a plant may have different cardinal points; hence fruits may suffer injury when vegetative parts escape unharmed, or flowers may be burned by temperatures that cause no injury to leaves.

Temperature is one of the factors in the climatic complex that plays a most important part in the natural growth and distribution of plants. In addition to being exposed to the vicissitudes of climate, crop or cultivated plants are subject to the intervention of man, and consequently they are frequently forced to attempt their life processes under uncongenial temperatures and other unfavorable environmental conditions.

The temperature limits for growth lie, in general, between 0 and 50°C. Some growth may take place in certain plants at, or even slightly below, the freezing point of water, while some fresh-water algæ that frequent hot springs may actually thrive at temperatures of 73°C. or slightly higher. Even our common crop plants show marked variations in their cardinal temperatures, as may be illustrated by the appended table of cardinal points of growth.

	Minimum, degrees Centigrade	Optimum, degrees Centigrade	Maximum, degrees Centigrade
White mustard (*Sinapis alba*)	0.0	21	28
Garden cress (*Lepidium sativum*)	1.8	21	28
Barley (*Hordeum sativum*)	5.0	28	37.7
Wheat (*Triticum vulgare*)	5.0	29	42.5
Corn (*Zea mays*)	9.4	33.7	46.2
Squash (*Cucurbita spp.*)	14.0	34	46.2

Types of Heat Injury.—The principal types of heat injury are (1) retarded growth and undersize or failure to mature the flowers and fruit; (2) localized killing of tissues or a sunburn or sun scald of leaves, flowers or fruits; (3) localized killing of stem tissues or the formation of heat cankers; (4) defoliation or premature shedding of leaves; (5) premature ripening of fruits; and (6) death of the plant as the result of a general heat necrosis. It should, of course, be recognized that the high degrees of heat are frequently accompanied by intense sunshine and extremes of drought which intensify the injury. Death of cells from high temperature results when there is an irreparable destruction of the molecular structure of the cytoplasmic body.

The dwarfing effect of high temperatures may be seen in the growth of our common garden asters when planted in the South. In regions where the summer temperatures average 95 to 100°F., they may produce an unbranched stem 6 or 10 inches high with a single flower not over an inch in diameter, as contrasted with the thrifty, branched growth with numerous chrysanthemum-like flowers that are normal in the more temperate climates of the North.

The writer is inclined to attribute certain wheat failures that have been observed in the Pacific Northwest to early seeding combined with abnormally high temperatures. It has been observed that under certain conditions, winter wheat that was seeded about July 15 to Aug. 1 either made a very poor growth and gave a reduced yield or failed almost entirely. Entire fields of such early seeded wheat have been noted when the plants that did come through the winter made a sickly, chlorotic, stunted growth and produced few or no heads. This permanent stunting

apparently results when the young seedlings are subjected to temperatures which fluctuate slightly above or below the maximum temperature for growth. The failure of winter wheat to head when planted in the spring is probably due to light rather than heat (see Chap. IX on Diseases Due to Unfavorable Light Relations).

During the periods of intense heat in the summer in the northern sections or frequently in the hot season in southern latitudes, there may be more or less burning of leaves, flowers or fruits. With leaves, there may be a death of marginal tissue, or internal necrotic areas may be formed, in many cases the effects resembling drought injury. Injuries of this type may result when the tissues are suffused with water and the atmosphere is humid, or very similar effects may follow when transpiration is greatly accelerated by low humidity and high temperatures (see Tip Burn of the Potato, p. 143). Somewhat similar injury of maples (Hartley and Merrill, 1915) and of cowpeas (Gibson, 1922) has been noted. The sun scald of beans described by McMillan has been attributed largely to light rather than to high temperatures (see Chap. IX on Diseases Due to Unfavorable Light Relations).

The appearance of sun-scald spots on the foliage of plants grown under glass is not uncommon. Isolated spots, lines of spots or dead streaks may be formed. These are attributed, in a large part, to the concentration of the heat by bubbles in the glass which act as burning lenses, and the lines of spots or streaks result from the shifting position of the sun. It has also been noted that water drops may act in a similar fashion as burning lenses, both in greenhouse cultures and in plants grown in the open. Spraying of delicate plant structures during the heat of the day may promote injury for the above reason and also from increased sensitiveness to heat due to a modified water relation. Heat cankers may result from high summer temperatures when trees have their trunks or limbs exposed as a result of clearings or cutting for roads or from any removal of shade, which has served as a protection. Bark cankers of this origin should not be confused with the so-called winter sun scald (see Chap. VIII).

The scalding of flowers frequently results from high temperatures. The browning of the rays of dahlia flowers in the early blossoms produced during the periods of high temperature and intense sunshine is a notable example. Perfect flowers are formed later in the season when lower temperatures prevail. Many fruits suffer from sun scald or sunburn, especially those which are succulent. Even fruits like the apple may be severely burned, during periods of intense summer heat, especially the sun-exposed face of fruits hanging on the outer branches. Injuries of this type may be intensified by lime sulphur or other sulphur spray (see Spray Injury) but may occur independent of any spraying operations. Strawberries, grapes and other fruits may frequently be scalded when

they are exposed to intense sunlight and high temperature following a humid period in which absorption was active, but transpiration checked. Sun-scald injuries are due not to the direct action of the air temperatures but to "excessive heat generated in exposed parts of plants by sunlight absorption" (Harvey, 1925). The sun scald of tomatoes is also a very common cause of complaint. Heat conditions which are not sufficiently intense to produce localized injury may accelerate the ripening processes and lower the keeping qualities of the fruits that are prematurely ripened.

Heat defoliation may occur in both deciduous and evergreen trees, and, according to various observers, the more exposed leaves or those occupying a peripheral position are less likely to be cast than those in a more protected position within the crown. Although leaves can reduce their temperature by an active transpiration, under certain conditions their temperature may rise above the maximum which their tissues can tolerate, and heat casting is the result. It is the belief that the inner leaves suffer first because of their greater sensitiveness to heat and also because the radiation of heat from them is retarded by the more exposed foliage. Seedlings of coniferous or deciduous trees or of herbaceous species may be seriously injured or even killed by high temperature, the injury being localized in the stem just above the ground level, thus resembling damping-off due to fungi (Hartley, 1918; Bates and Roesner, 1924). This trouble has been called *white spot*, especially on conifers. The order of heat tolerance for four evergreen seedlings was as follows: lodgepole pine, yellow pine, spruce and Douglas fir. It was suggested that temperature may be the critical factor which limits or prevents natural reproduction (Bates and Roesner, 1924). Similar heat injury has been described for cowpeas, rye, buckwheat, beans, oaks, maple and vetch. Severe injury to cucumber seedlings was observed by the writer when heat was the only factor that seemed responsible. It seems probable that heat injury to young tender seedlings is much more common than recorded experiences would indicate. Severe injury of this type to a field crop is well illustrated by the heat canker of flax (see special treatment).

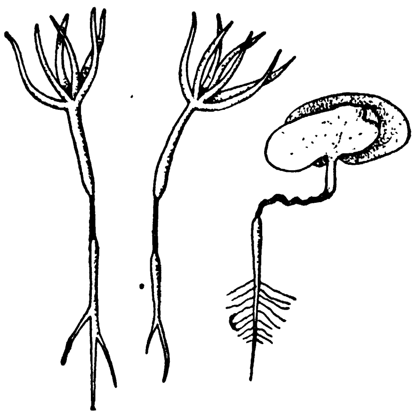

FIG. 47.—Heat canker of pine and beech seedlings. (*Redrawn after Münch.*)

Not only seedlings but mature herbaceous plants may succumb from high temperatures during the intense heat of southern summers. A single illustration will emphasize this behavior. The common garden nasturtium will thrive and flower luxuriantly in the early part of the grow-

ing season in middle Texas but suffers from "sunstroke" during the midsummer period of cloudless skies and temperatures ranging from 95 to 100°F. or more.

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TIP BURN OF THE POTATO

This disease of the potato is characterized by burning or browning of the tip and margins of the leaflets under the influence of excessive heat and sunshine. It is only in recent years that this physiological disturbance has been differentiated from the "hopper burn," a trouble which appears to be connected with the work of leaf hoppers.

History.—A disease of the potato was described as "tip burn" by Jones (1895), who characterized it as showing the death of the "leaves at their tips and margins, which portions dry, blacken and roll up or break off." This disease was common in Vermont in 1894 and 1895 and was also sufficiently in evidence in Connecticut, Michigan and Wisconsin (Goff, 1896) to attract the attention of experiment-station workers. From that time on, the disease was frequently mentioned in general considerations of potato diseases, but no detailed studies of the trouble were made until the work of Lutman (1919). During the same year (1919), Ball published work which pointed to leaf hoppers as the cause of the burning of the leaves and therefore designated the trouble as "hopper burn." The present recognition of two similar types of troubles, the one due to a physiological disturbance, the other to the work of leaf hoppers, makes it difficult to say to what extent the two diseases were present when the earlier studies were being made. Entomologists have been rather inclined to the belief that all tip burn is "hopper burn," but Lutman (1923) believes that many of the earlier studies undoubtedly dealt only with the physiological form and that hopper burn is a later development. A detailed study of the relation of the water pores and stomata of the potato leaf to the early stages and advance of the tip burn has been made by Lutman (1922), and he has also pointed out the differences between true tip burn and hopper burn (1923).

Symptoms and Effects.—Tip burn is first in evidence as a slight wilting and yellowing of the tissues at the extreme tips of potato leaflets or, more rarely, at the margin at some point back of the tip as well as at the tip. This yellowing is soon replaced by a browning and death of the tissues, and the dead area is extended from the tip downward or from the margin inward until in extreme cases entire leaflets are brown and dead. The browning begins at the tip or margin and gradually advances downward or inward into the pale-green or chlorotic tissue. Under favorable conditions for the development of the disease, the lesions may gradually advance, or the progress may be delayed or checked by the return of better growing conditions. The amount of tip burn varies

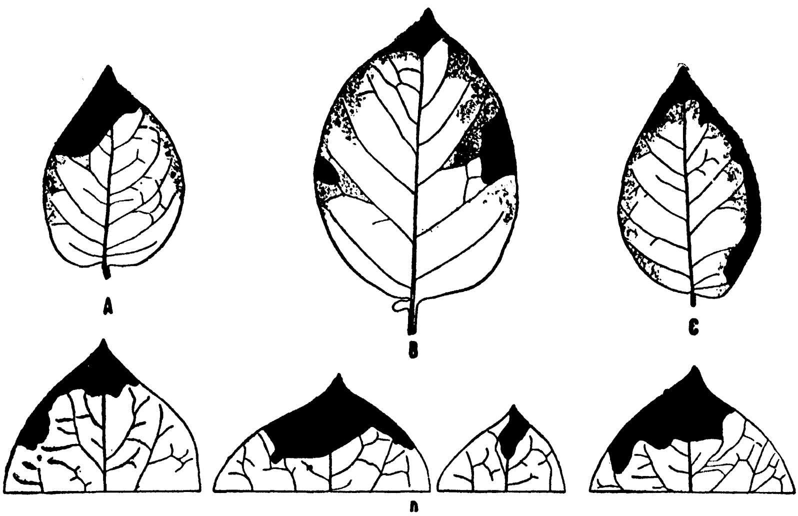

FIG. 48.—Typical tip-burn injuries. Leaflets *A*, *B* and *C* were more or less rolled; leaflets marked *D* were not rolled to any extent. (*After Lutman, Vt. Bul.* **214**.)

with the position of the leaves and is also influenced by the age and maturity of the leaves. Young leaves which are standing nearly upright appear to suffer the least, while the tip blight is more severe on those leaves which are older and occupy a position so that the sun strikes them more nearly at right angles to their surface.

The older leaves droop so that only the tips of the end leaflets are exposed, but those of all of the leaflets may hang down and be struck by the tip burn. The oldest leaves are likely to be under the shade of those from the middle of the stem and so suffer less than those in the middle. If they are exposed at all, they succumb readily, and all of the leaflets die in a few days. The earlier attacks, however, are likely to occur on the middle leaves, and after they have become thoroughly scorched and dead the oldest leaves are exposed and in turn succumb (Lutman, 1919).

Tip burn appears to reach its maximum severity in the New England states in late July or early August. Lutman records the killing of

approximately 40 per cent of the foliage on certain plants by Aug. 17, in a typical outbreak of the disease in Vermont in 1917. This injury resulted during the preceding period of July 20 to Aug. 17. The physiological effect of this early loss of foliage must be apparent. The growing season is cut short, and consequently the yield will be correspondingly reduced. The losses are variable with the season and in some environments may be negligible, but the idea has been expressed that in the lower acre production of potatoes in the United States, as a whole, over that of Germany and England, the tip burn plays a part next in importance to late blight and the Colorado potato beetle.

Since tip burn and hopper burn may be working independently or simultaneously in a given environment, the distinguishing marks of the two troubles may be contrasted: (1) In hopper burn, the vascular system leading from the midrib is the center of the disturbance, and the death of tissues advances outward, while in tip burn the death of tissues advances from the tip or margin inward. (2) Hopper burn is not restricted to any part of the leaf but may involve any portion, while the tip burn always starts at the extreme tip of a leaflet or, more rarely, at the margin also. In hopper burn, characteristic *V*-shaped areas of dead tissue with the broad end marginal frequently result, a main lateral vein marking the center of each.

Etiology.—Previous to the discovery of hopper burn, there seems to have been a general agreement that tip burn of the potato was due to shortage of water during the hot periods of midsummer. In 1895, Jones stated:

> It is attributable to unfavorable conditions surrounding the plant, especially to the hot, dry weather with insufficient water supply. It is aggravated by any other conditions which may tend to lower the general vigor of the plant, such as insufficient food supply, attacks of insects and the early blight fungus. This difficulty has not been observed to any serious degree upon plants until after they pass the blossoming period and naturally begin to weaken.

It has been pointed out by Lutman (1919) that the principal factors operative in producing tip burn are *heat and the intensity of light.* The disease seems to reach its greatest severity at the crest of heat waves or high-temperature periods, and it is significant that such periods are generally marked by minimum humidity and very intense sunshine. The potato makes its best growth during long, relatively cool, equable summers and in the South must be grown before the advent of the intense summer temperatures (Orton, 1913). In many portions of the southern United States, potatoes suffer with tip burn and ripen prematurely if planted too late or if the hot weather comes too early. This heat burning of foliage is not uncommon in the South on various plants and is noticeable even in certain species of the native vegetation, especially in

the lighter soils. The fact that temperature and light are more important than water shortage in producing the trouble can be noted by the behavior of plants which are not suffering from water shortage.

It has been possible to produce tip burn under artificial conditions, the symptoms and effects agreeing in the main with the development of the disease under natural conditions.

Sunlight may act either chemically to cause the destruction of important leaf constituents, *e.g.*, chlorophyll, or it may so warm the leaf as greatly to accelerate the water loss (Lutman, 1919).

If we think of transpiration or water loss as more than mere evaporation—an active process dependent upon the chemical and physical properties of the living substance—the influence of light and heat in accelerating the water loss may be appreciated. It seems probable that these changes take place and that the excessive water loss follows. The cells of the affected areas lose water until they are plasmolyzed or lose their turgidity and, when the changes reach a certain degree of intensity, recovery is impossible and the cells die. The parts which heat and light play in the production of tip burn have been demonstrated by shading, since it has been possible to prevent or reduce the disease by the use of muslin or cheesecloth screens.

The greater freedom of the young leaves from tip burn is explained in two ways: (1) by their upright position, which causes the sunlight to fall more nearly parallel to their surfaces and thus gives them a measure of protection; and (2) by the higher concentration of the cell sap over that in the lower leaves, which retards the loss of water. It has also been pointed out that plants in the early part of the growing season have a higher sap concentration in the tissues of the foliage than elsewhere but that with the oncoming or passage of the blossom stage the stalk tissues have the highest osmotic pressure, especially during periods of low humidity and high temperatures. It seems probable that under such conditions, the cells of the stalk may draw upon the leaves as well as the roots for water. This will explain, in part at least, the greater sensitiveness of the potato plant after it has reached the blossom stage.

On cloudy, humid days, the pressure in the stalks tends to subside, and a nearer approach to equilibrium becomes established between stalk and foliage. The advance of the tip burn then stops (Lutman, 1919).

A special study has been made of the relation of water pores or hydathodes to the inception and advance of tip burn, and the following conclusions have been presented:

1. Potato leaves are provided with hydathodes around their margins but especially towards the tip. These hydathodes resemble the stomata but are placed so as to open directly on the vessels of a large vein which runs close to the

margin of all the leaflets. A group of these hydathodes is located just at the tip of each leaflet where many of the vessels of the marginal vein end.

2. Tip burn begins beneath these hydathodes, especially at the tip of the leaflets. The death of the tissues under the terminal water-pore group leads to the browning and shriveling of the extreme tip of each leaflet. The tissues die under the water pores along the side of the leaflet and apparently break the continuity of the water-supply system of the marginal vein.

3. The further advance of tip burn into the leaf is due to direct sunlight acting on the cells of the leaf leading to such an extreme plasmolysis that the cells are not able to recover. Associated with this plasmolysis is the destructive

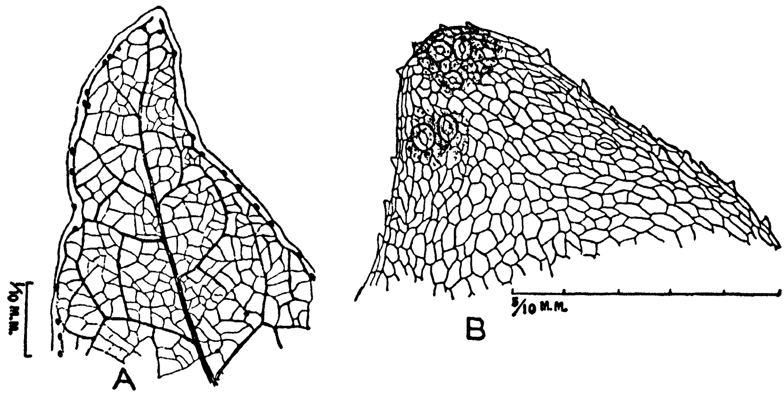

FIG. 49.—*A*, tip of very young leaflet showing venation and the position of the hydathodes; *B*, tip of mature leaflet in surface view, showing groups of hydathodes. Dead tissue under the pores is indicated by dotting. (*After Lutman.*)

action of the sunlight upon the chlorophyll bodies and the chlorophyll itself. This expresses itself in the yellowing of the green portion of the leaf in the region dead from tip burn (Lutman, 1922).

At this point, it may be well to note that the etiology of the so-called "hopper burn" is not well understood. Ball (1919) reported it to be specific, *i.e.*, caused by a single species of leaf hopper (*Empoasca mali* Le B.) but not by other closely related species, and advanced the idea that hopper burn was an insect-transmitted trouble, somewhat similar to curly top of sugar beets. In 1922, Eyer reported that he had been able to produce hopper burn by injecting an extract of leaf hoppers, especially of the nymphs, into healthy plants and that the "specific" from diseased plants may be reinoculated into healthy plants and again produce the characteristic burning. Lutman (1923) is of the opinion that his evidence is not conclusive and advances the theory that the leaf hopper, in sucking the juice from the veins,

. . . removes the water from the tubes and from the adjacent sieve tubes, companion cells and elongated parenchyma to such a degree that they plasmolyze and collapse to a point where they cannot regain their turgidity, and, as a consequence, they die and become brown in color.

If this is true, it would not be necessary to assume the injection of a specific "toxin" or an inciting agent.

Varietal Susceptibility.—Different varieties show varying degrees of susceptibility to tip burn. In general, it may be stated that

> Early varieties are affected sooner and more seriously than later ones. The foliage of the latter often largely survives the weather producing tip burn and renews growth in September (Lutman, 1919).

On the basis of studies in Vermont in 1915, Irish Cobbler, Boree, White Ohio and Triumph showed 100 per cent, and Early Ohio and Early Rose 90 per cent of the foliage dead from tip burn on Aug. 23, while 15 other varieties under the same conditions showed from 5 to 30 per cent of the foliage dead. Very similar variety relations were noted during the season of 1916. The general observation has been made that potatoes suffer much more from tip burn on light, sandy soils than on the heavier soils, and a low potash content of the soil also seems to aggravate the trouble.

It is of interest to note that other crop plants show injuries similar to the tip burn of the potato during very hot summers. This is true of lettuce when grown either in the open or under glass. Even the regions of commercial production of head lettuce may have seasons that cause much tip burn, and in many such cases the injured leaves offer an easy center of origin for slimy, soft rot which may cause heavy losses in transit to market.

Control.—Since tip burn is the result of intense light and high temperatures combined with low humidity and frequently of water shortage in the soil, it cannot be entirely prevented in those regions in which the exciting factors are likely to be operative. The trouble may be reduced by giving attention to the following: (1) the selection of late varieties if experience shows that the early varieties suffer; (2) the avoidance of the lighter soils of an environment which may predispose to the disease; and (3) spraying to control flea beetles, leaf hoppers and other insects. Spraying with Bordeaux has been reported to give a protective action in the case of physiological tip burn alone, due possibly to a shading effect or to a retardation of transpiration. The use of Bordeaux as a repellent for leaf hoppers has been recommended as the only practical method for the prevention of "hopper burn" (Dudley and Wilson, 1921; Parks and Clayton, 1923).

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HEAT CANKER OF FLAX

Flax plants may be injured in such a way as to cause them to break over at or near the ground line and are then said to be affected with flax canker. Various factors are responsible for this behavior. A definite fungous canker, in the nature of a damping-off disease, due to *Colletotrichum lini* Bolley, has been recognized in America and also in other parts of the world, but it seems to be definitely established that another type of wide occurrence is of a non-parasitic character and due to high temperatures. For this reason, the name of "heat canker of flax" has been proposed. As a result of investigations carried on since 1916, it has been shown that the Colletotrichum canker is rather rare in the United States during some years and, when present, affects seedlings in the main, while heat canker occurs "somewhat uniformly in the northern Great Plains area and causes a marked loss in flax production."

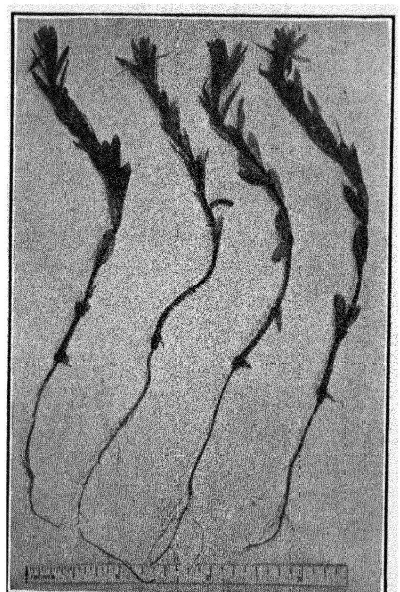

Fig. 50.—Flax seedlings affected with heat canker. (*After Reddy and Brentzel, U. S. Dept. Agr. Bul.* **1120**.)

Symptoms and Effects.—Heat canker is generally first noticed by plants breaking over at or near the ground level "as though whipped off by the winds or gnawed by insects." This effect is due to the killing of the cortex of the stem above the ground line, while the plants are still young and tender.

Generally speaking, if the injury occurs when the plants are less than 3 inches in height, the tissues collapse at the point of injury and the plants wither and die.

If the injury occurs somewhat later, when the plants are 3 to 5 inches in height, only the cortex is killed, allowing the plants to topple over but usually to remain alive for days or weeks because of the uninjured vascular system within. Only in rare instances are plants more than 5 inches in height injured in this way. Numerous more mature specimens of heat-cankered flax can be found, but in such cases growth continues after the initial injury. Enlargement of the stem occurs just above and sometimes just below the injury. In most cankered plants, the stem is severed, sooner or later, at the point of girdling by the winds or by the disintegration of the remaining tissues, due to the action of saprophytic organisms. Otherwise the plant dies when the starving roots can no longer support the increasing needs of the aerial portion (Reddy and Brentzel, 1922).

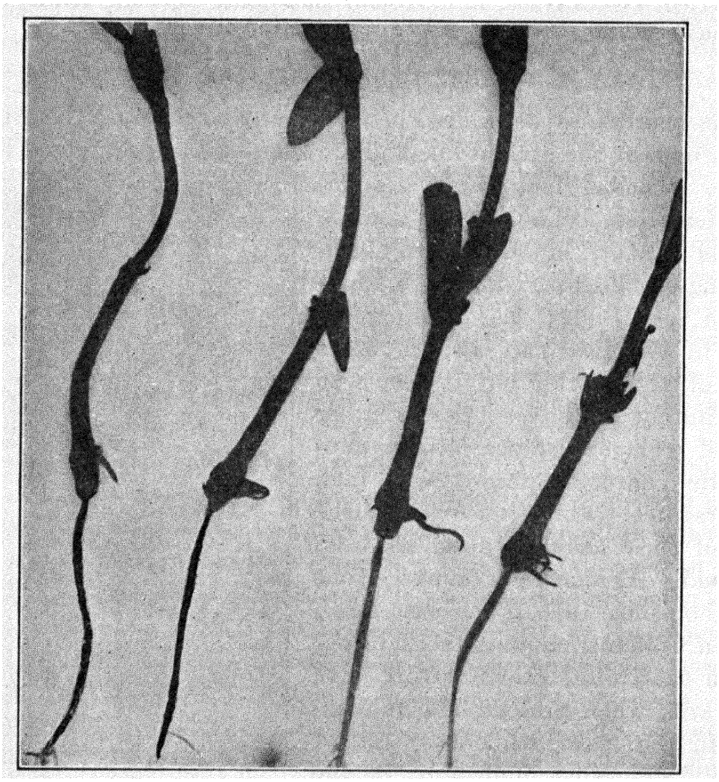

FIG. 51.—Basal portion of plants shown in Fig. 49 enlarged to show constricted areas and enlargements. (*After Reddy and Brentzel, U. S. Dept. Agr. Bul.* **1120.**)

The marked constriction of the stem within the limits of the canker is due to the death and shrinkage of the cells of the cortex, while the enlargement above the canker is due to the interruption of the downward movement of elaborated food which can no longer be carried to the root system. In case the enlarged portion of the stem comes into contact with moist soil, adventitious roots may start, but under dry conditions they do not form. A somewhat similar form of trouble occurs in the drier areas of the Great Plains later in the season.

Losses have been caused by the heat canker varying from slight to heavy, with almost complete destruction of fields in isolated cases. In the experimental plots at Fargo, N. D., the percentage of cankered plants when unshaded ranged from 37.2 to 46.

Etiology.—The heat canker of flax is the result of the high temperatures of surface layers of dry soil that are in immediate contact with the tender tissues of succulent young stems. The amount of the injury is apparently influenced by the compactness of the soil, the succulence of the tissues and the absolute temperature. In the heat-canker plats (Fargo) during 1920 and 1921, there were many days when the maximum soil temperature at a depth of ½ inch varied from 40 to 50°C. (104 to 122°F.), while the temperature at the surface was in many cases 4.5 to 7°C. higher.

The first evidence of the non-parasitic nature of the disease was obtained by making numerous cultures from typical cankers. No one organism was constantly present, none that was isolated was ever capable of producing the disease when used in inoculations and in many attempts at isolation no organisms appeared. Field observations and tests in experimental plats gave evidence in favor of heat and light being the important causal agents. Some of these relations were as follows: (1) Canker was more severe in rows with wide than with close spacing and also more destructive in thin than in thick sowings; (2) a growth of weeds seemed to lessen the amount of the disease; (3) the shading by a cereal nurse crop had the same effect in reducing the canker as a weed crop; (4) providing partial canvas shade reduced the disease or prevented it entirely; (5) the canker was less when the soil was covered with a yellow sand to a depth of ¼ inch than on the same heavy, dark soil unprotected by the sand layer; (6) a firm crusted surface soil seemed to favor the disease, while a mellow soil without a crust lessened it in amount; and (7) a cankered condition similar to heat canker was produced artificially.

Heat canker always appeared following the days when the temperature at the surface of the soil was high. The observations indicated "that under conditions favorable for the production of heat canker, the critical temperature is about 54°C." When moisture and temperature conditions produce a soft, succulent growth of the young plants and this is followed by crusting of the surface and a period of critical temperatures, the heat canker is likely to develop, while the same temperatures will have a less injurious effect on plants which have made a firmer growth. Early seedings sometimes escape the trouble because they may pass the susceptible stage before the advent of high temperatures.

The soil crust caused by rains brings the surface soil in immediate contact with the tender surfaces of the succulent young flax stems. Injury results when such surface layers in immediate contact with the tender living tissues reach the high temperatures. This surface crust may act as a conductor of heat to the

plant . . . The evidence, therefore, indicates that heat canker of flax results from a combination of succulence in young plants and high temperatures of the surface soil in immediate contact with such succulent tissues (Reddy and Brentzel, 1922).

Prevention.—Since the trouble is due to the high temperatures during the early period of growth, heat canker should be controlled either by seeding early, so that the seedlings pass the susceptible stage before the advent of the hot weather, or by adopting cultural practices that will partially shade the young plants and thus hold down the temperature of the surface soil. For this latter purpose, higher rates of seeding and drilling north and south rather than east and west have been suggested.

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CHAPTER VIII

DISEASES DUE TO LOW TEMPERATURES

Living plants, either growing or dormant, or plant products are likely to be subjected to temperatures sufficiently low to cause either death or injury. The character and extent of the injury will vary with the temperature and the condition of the plant or plant structures.

General Effects of Low Temperatures.—In discussing the general effect of temperature upon growth, it was pointed out that for each species, variety or strain there is a certain degree of warmth, the *optimum* at which growth is most rapid. As the temperature sinks below this optimum, growth becomes less and less rapid and finally ceases at the *minimum*. This retardation or checking of growth is the inevitable result of low temperature. A second effect of low temperature is the prevention of chlorophyll formation or the slower construction of this pigment, with the result that parts normally green may become yellow. In some plants or plant parts, cold causes the development of red pigment, which apparently obscures the lesser degree of chlorophyll development. When the temperature sinks to a sufficient degree, freezing of plant tissue results, and death may follow, or with the rise of temperature, the frozen tissues may thaw out without any appreciable injury.

The final result following exposure to low temperature will be variable, depending upon specific peculiarities of plants, moisture relations, length of exposure, degree of cold and other internal or external factors. It may suffice to say that the final results in the action of cold will fall into three groups: (1) recovery or return to normal with the advent of temperatures favorable to growth; (2) either the parts may be lost, malformed or disfigured or, in annuals, the entire plant may become a permanently deformed cripple for the rest of the growing season or, in perennials, may lead a struggling existence in a condition of lowered vitality for a period and finally attain normality, or the derangement may become more pronounced and end in death; and (3) the injury may be of the *acute* type in which sudden death of the plant is the outcome.

How Freezing Causes Injury.—There has been much discussion as to how freezing causes the death of plant cells. The early theory of cell rupture due to expansion of the cell sap was soon discarded, because it was shown that water was withdrawn from the cells into the intercellular spaces where the freezing occurred. It is now generally agreed, however, that this withdrawal of the water from cells is the serious feature in

freezing injury. Several different theories for the injury have been offered.

1. *Cold Death by Poisoning.*—The withdrawal of water is thought to concentrate the cell sap so as to leave substance actually toxic to the protoplasm (Lindforss, 1907).

2. *Mechanical Injury.*—With this concept, death results from "mechanical injury of the protoplasm caused by the compression of the ice crystals which accumulate in the intercellular spaces" (Maximov, 1929).

3. *Destruction of Protoplasmic Structure or Architecture.*—The loss of the semipermeable character of the protoplasm permits the water to pass into the intercellular spaces. If this structure is permanently lost, death results and the intercellular moisture which cannot be reabsorbed is soon lost by evaporation (Müller-Thurgau, 1880). This third theory has been modified by Stiles (1930), who has presented evidence to show that the protoplasmic changes and the consequent death of the cell result "from the formation in the protoplasm of relatively large ice crystals and the consequent alterations of the space relations of the phases constituting the colloidal complex of the protoplasm." Recovery following freezing would take place only if thawing permitted the regaining of the original space relations.

Variation in Cold Resistance or Hardiness.—Plants show wide variation in their tolerance of low temperatures. It is well known that tomatoes, potatoes, beans, cucurbits, corn, dahlias, etc., are extremely frost sensitive, generally suffering acute injury with the first formation of ice crystals. Other annual plants, such as spinach, lettuce, the various cereals, etc., are much more frost resistant, and in certain regions hardy varieties may even behave as winter annuals, beginning their growth in the fall of one season and completing it during the next. Contrast, for example, the difference in the frost resistance of dahlia roots or potato tubers and the bulbs of such hardy plants as tulips, narcissuses, jonquils, etc. In general, the crops of our temperate regions which have developed the biennial habit, such as carrots, beets, parsnips, salsify, cabbage, etc., are frost resistant. Some frost-sensitive plants which behave as annuals in temperate regions may become perennial in the moderate climate of southern regions. The perennials exhibit all gradations from tender to extremes of winter hardiness, and one of the tasks of the plant breeder is the production of hardy varieties of farm crops, fruit plants and other desirable plants which will extend the range of profitable production.

The Basis of Hardiness.—Many different theories have been offered to explain hardiness or resistance to injury from freezing. The most probable are based on the Müller-Thurgau theory of death by water loss incident to the destruction of the protoplasmic structure. Since water

retention is the basis of hardiness, the main factors which are concerned in water retention may be noted:

1. *The Property of the Hydrophylic Colloids of the Cell to Hold Water by Such Phenomena as Adsorption, Adhesion or Molecular Capillarity.*—Water may be *unfree* or *bound, i.e.*, not easily freezable, or *free*, in which state it easily freezes. This "bound" water may be the total water of hydration of all the substances in the plant, but water bound by other than colloids is thought to be of minor importance. The imbibition force of the cell colloids is a generally recognized principle. Some workers have attached special importance to the *pentosans* as the organic compounds mostly responsible for the water-retaining power of the colloidal complex. There can be no doubt that they play a part, but variable conclusions have been reached by different investigators, some reporting a marked parallelism between pentosan content and hardiness while others report no quantitative relation.

2. *The Carbohydrate Content.*—A sufficient number of cases have been recorded showing higher total carbohydrates in hardy than in tender varieties to attach some importance to this quality. It has been shown that sugars have the ability to protect proteins from precipitation on freezing and that there is an increase in sugar in many plants when exposed to low temperatures.

3. *The Osmotic Concentration Due to Various Solutes or Soluble Compounds in the Cell Sap of the Vacuoles.*—The concentration of the sap may play a part in delaying inception of ice-crystal formation, but it appears to be an accompaniment of hardiness rather than the real cause. Neither high carbohydrate content nor high osmotic value of the cell sap is a constant accompaniment of hardiness.

Attempts have been made to correlate hardiness with other measureable factors, *e.g.*, quantity of press juice, moisture content, total solids, amino acids, organic nitrogen or viscosity, but none has proved to be an infallible indicator. The only recognized reliable test of hardiness is artificial freezing at controlled temperatures with the determination of injury.

The recent summary statement by Stiles (1930) appears to express the present concept in excellent form:

> In cases of frost-resistant plants, however, it is probable that water is bound to hydrophile colloids of the protoplasm and is non-freezable, so that the formation of ice crystals and the consequences of their formation to which the death of the cells is attributed do not take place.

Frost Injury and Winter Injury.—For purposes of convenience, low-temperature injuries may be considered under the heads of *frost injury*, or injuries which result from low temperatures after plants have started into growth in the spring, during the period of vegetative activity or before

they have matured and entered their period of winter dormancy, and *winter injury*, or injuries that result from low temperatures after the end of the growing season or before growth starts in the spring. Frost injury involves mainly frost-sensitive annuals or the foliage, blossoms and young fruit of perennial plants, while in winter injury we may be concerned with winter annuals, biennials or herbaceous perennials which retreat underground or woody plants which normally pass the winter in a dormant condition, either with or without the shedding of their leaves

FROST INJURY

Low-temperature Injury to Leaves or Young Shoots.—In many plants, the temperature at which chlorophyll ceases to be formed is higher

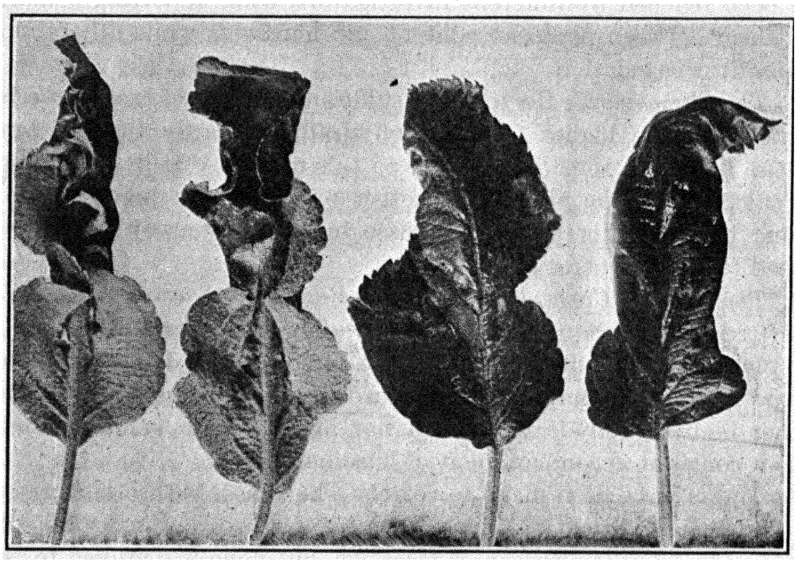


FIG. 52.—Frost curling of apple leaves.

than the minimum for growth, so that plants suffering from chlorosis due to cold may still continue to grow slowly. The yellow color of early spring as noted in overwintering rosettes, young seedlings of spring annuals or the tips of the first shoots or leaves from herbaceous perennials is the result of temperatures unfavorable for chlorophyll formation. With the advent of warm weather, the chorotic structures will become green if the chloroplasts have not been seriously damaged. Temperatures not sufficiently low to have a killing effect upon the tissue may cause a clumping or disorganization of the chloroplasts, and normal greening may not occur even with favorable temperatures. Such affected leaves may persist through the life of the plant, or they may blight before the end of the growing season. The pronounced yellowing of winter barley in the early spring is not an uncommon phenomenon. Low temperatures

in connection with the saturation of the soil with water, which interferes with normal root activities, are the inciting factors. In some plants, the temperatures which are unfavorable for chlorophyll formation induce the formation of a red pigment, anthocyanin, which is dissolved in the cell sap, and moderate or intense reddening of the leaves is the result. In certain species of trees or shrubs, the first spring foliage may be highly colored with red, but this fades out with the arrival of warm weather. Certain varieties of winter wheat, if examined in the early spring, will show considerable reddening of the leaves, while in certain sections oats may show what has sometimes been called the "red leaf disease." The

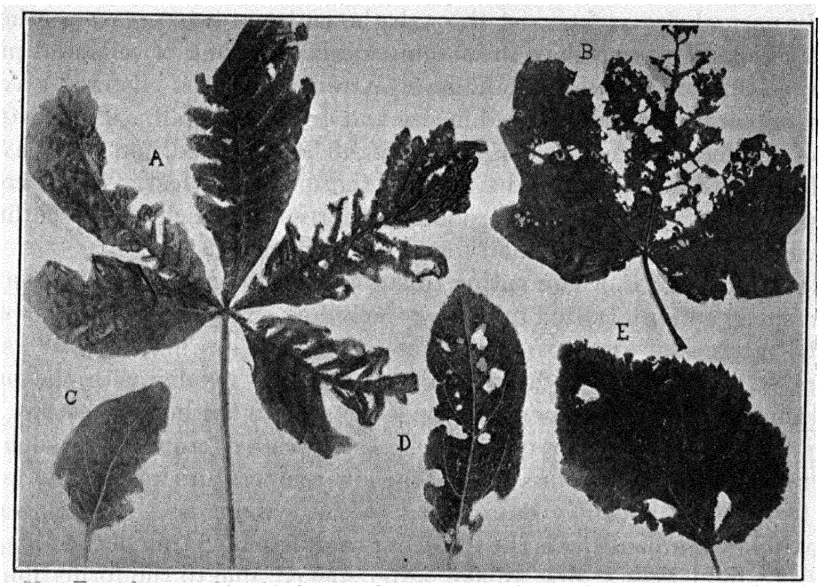


FIG. 53.—Frost-lacerated and deformed leaves. *A*, horse-chestnut; *B*, maple; *C*, lilac; *D*, apple; *E*, linden.

autumnal coloration characteristic of senility in deciduous leaves is an accompaniment of internal changes leading to the transfer of plastic materials into the twigs or branches before the final leaf fall or the advent of killing frosts. Temperatures above freezing have been shown (Sellschop and Salmon, 1928), to cause injury especially to certain tender species, the responses including chlorosis, blistering, wilting, localized, or general necrosis, with death resulting to the more sensitive species. Rice, velvet beans, cowpeas and cotton were killed by an exposure of 60 hours to 0.5 to 5°C., while potatoes, sunflowers, tomatoes and flax were not injured by the same exposure. Various gradations of sensitiveness occurred between these two extremes.

The action of low temperatures on young leaves just unfolding from the bud may cause much crinkling, puckering or curling of leaf blades. This effect is very common in the apple orchards of the Pacific Northwest,

and leaves showing these abnormalities are sometimes so similar in external appearance to those of peach leaf curl that orchardists are often led to believe that an infectious disease is prevalent. Leaves affected in this way are said to be "frost blistered," since the lower epidermis is raised in places from the underlying mesophyll. The epidermis of these blistered areas may crack later, thus exposing the interior chlorenchyma, many of the cells of which may become more or less filamentous due to the release of the normal pressure of the lower epidermis. The Jonathan apple, for example, is very sensitive to frost blistering of the leaves, and all gradations of injury may occur. Slightly affected leaves may reach nearly normal size and persist through the entire growing season, others more seriously injured may show some localized killing of cells and premature fall is likely, while in the most extreme cases the blistered leaves may remain very much reduced in size and dry up and fall. The blistering of the leaves is not so serious as might be supposed, for new leaves are formed which will take the place of those that are lost. A fair crop of apples may sometimes be produced by trees on which all of the first leaves show the most extreme type of frost blistering.

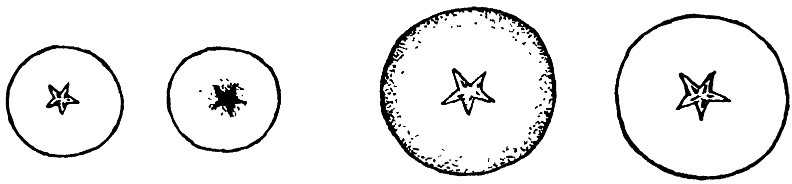
In some seasons of late spring frosts, young leaves of some trees and shrubs may suffer localized injury by the killing or splitting of the tissues in the intercostal areas, so that later the leaf blades may appear as if lacerated or torn. This laceration may be quite regular with the production of comb-like segments of the leaflets, as in the horse-chestnut, or more irregular, as in lilacs or maples. In the case of stone fruits, such as cherries, plums, peaches or apricots, the low-temperature injury may take the form of an irregular shot holing of the leaf blades. The extreme effect of low temperature is the partial or complete blighting of the foliage. The affected leaves at first appear water soaked, due to the formation of the ice crystals in the intercellular spaces, and with the melting of the ice remain limp and flaccid, without any return of turgidity. The affected tissues become rapidly discolored and with the evaporation of the moisture become shriveled, curled and brittle and soon fall or weather away. It is worthy of note that all leaves of even frost-sensitive plants are not equally susceptible to death by freezing. Two bean plants, for example, growing side by side in the same hill may have the leaves of one killed while those of the other suffer but little injury.

Young shoots together with the leaves they carry may be killed back either completely or partially by spring freezes. This would apply to herbaceous perennials like alfalfa or to woody species of evergreens like spruces or firs. In such cases, the drooping blighted shoots may suggest a bacterial or fungous invasion. Severe damage of this character may be encountered in evergreen nursery stock.

Injury to Blossoms and Young Fruits.—The blossom buds, blossoms or young fruits of our tree fruits or other perennials which are developed

in the early spring are frequently subjected to critical temperatures which cause a blighting of buds or blossoms and a consequent failure to set fruit, or, later, frosts may affect fruit that has already set and cause it either to drop or to be malformed.

The "danger point" for the blossom buds, open blossoms and setting fruits of pome and stone fruits shows a range of 5 to 6°F. The blossom buds that are closed but showing color are more resistant than the open blossoms, while the setting fruits are still more sensitive. The killing temperatures according to various authorities vary for the different species and also within the species: (1) for closed buds that are showing color, 20 to 30°F.; (2) for open blossoms, 25 to 30°F.; and (3) for setting



A *B*

FIG. 54.—Diagrams of sections of normal and frost-injured apples. *A*, soon after the petals have fallen, showing central necrosis; *B*, when ¾ to 1 inch in diameter, showing peripheral necrosis.

fruit, 27 to 32°F. In some localities, the frequency of late spring frosts makes the production of certain crops unprofitable and uncertain. Even in the recognized fruit regions, destructive frosts may occasionally cause almost complete losses, and experience must be the guide as to whether their frequency is sufficient to justify orchard heating or other frost-protective measures.

The essential organs of the flower, the stamens and pistils, are generally more sensitive than the accessory parts. Injury by slight freezing may be marked by a discoloration of the pistil, which becomes brown or black, while surrounding parts show but little deviation from the normal, but at lower temperature there may be a complete blighting of all the flower parts. The "black eyes" of strawberry blossoms which have suffered a knock-out blow by Jack Frost are typical of frost injury. The achenes and receptacle turn brown or black, while the petals remain unaffected. Young apples which have already set at the time of the killing frost may not show any external evidence of injury, but, if the fruits are cut across, the centers may appear brown or almost black, due to the killing of the rudiments of the seed and some adjacent tissue. A high percentage of young fruits showing such internal discoloration of the seed cavities fails to develop further, and these fruits are soon shed, while others less severely injured may make some growth and fall later, while still others may reach maturity but be undersized, irregular or one-sided with sterile or seedless core cells. In some cases, the severe temperatures

are not experienced until the fruit is ½ inch or more in diameter, and then the peripheral tissues may suffer more than the young seeds or the tissue adjacent to the core cavity. Most of the fruits showing this external injury fail to mature, but some continue their growth and show more or less internal necrosis of the pulp. This effect was not uncommon in Washington following a late freeze in 1920.

Even a crop like wheat or rye may be injured by late spring freezes which occur about the time the young heads are emerging from the boot or when they are in blossom. Heavy damage occurred in winter wheat in certain sections of the Inland Empire of the Pacific Northwest, during the late freeze of 1919. In the most severe injury, there was a complete

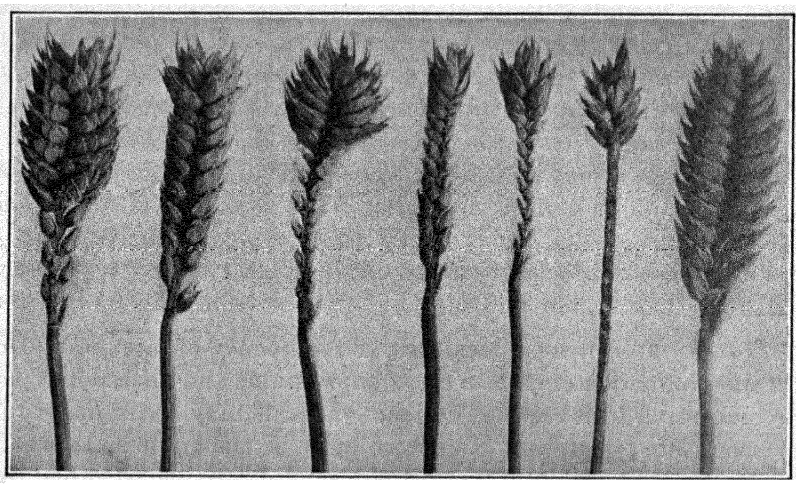


FIG. 55.—One normal and several frost-injured heads of club wheat. (*Photograph by B. F. Dana.*)

sterility of the heads, with a complete loss of the crop, while in other cases the freeze caused partial sterility only. Partial sterility due to late frosts is quite noticeable in some of the club wheats, the sterile florets occupying either a basal or, more rarely, an intermediate position. Frost injury to the culms may accompany inflorescence injury or occur independently. As a result of freezing, the meristematic tissue at the base of certain internodes may be killed, and the culms break over later at the injured point. If such bent or lodged stems are examined, the base of the affected internode will be found to be shriveled and frequently somewhat discolored. While upper nodes may be affected in this way, it seems probable that freezing of basal internodes in contact with the soil may cause similar injury and that such weakened tissues may offer little resistance to the inroads of soil-infesting fungi.

Frost Russeting of Orchard Fruits.—Young fruits that are not killed by the action of frost may be disfigured or malformed. Russeting, or the formation of brown, rough areas on the skin that should otherwise be

perfectly smooth, is one of the recognized effects of low temperatures during the early stages of growth. It should, however, be recognized that complete or partial russeting of the skin is a normal character in certain varieties of both apples and pears and that other factors besides low temperature may bring about russeting. Russeting is due to localized injury to the surface cells and the formation of cork cells beneath, which rupture the surface and cause the brown, rough skin. Frost russeting may occur in the form of a ring or band extending completely around the middle of a fruit. The retarded growth beneath the russeted

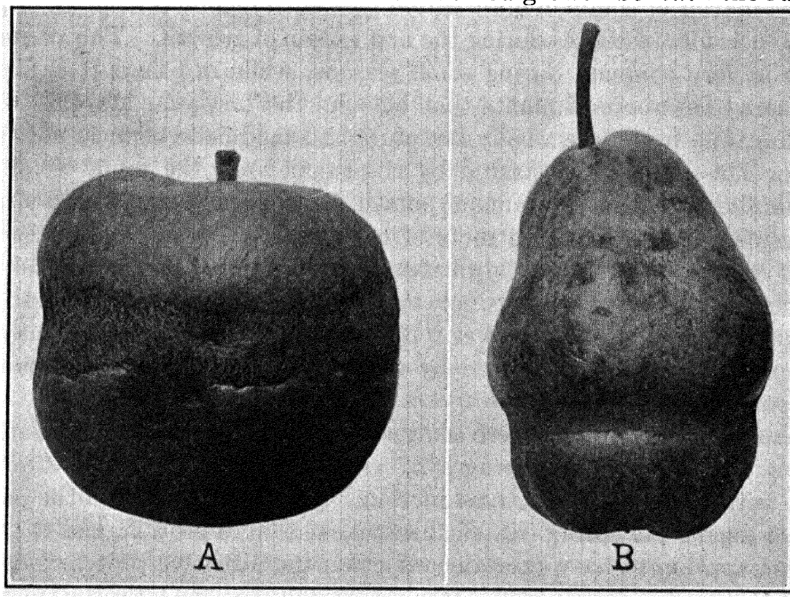


FIG. 56.—"Belted fruits." *A*, apple; *B*, pear.

band causes a slight constriction. Such "belted or banded" fruits are not uncommon in apples and pears. In other cases, probably less frequently, russet rings, ½ inch or slightly more in diameter, may appear on the cheek of an apple, the russeted tissue being sharply marked off from the healthy tissue occupying the centers but gradually fading out around the periphery. In some cases, large irregular patches of russet skin may develop at either the calyx or the stem end of a fruit, while scattered or diffuse russeting may characterize the more moderate types of frost injury to the skin of fruits.

Frost Injury of Sensitive Annuals.—It is a matter of common observation that frost-sensitive annuals frequently suffer acute injury from spring freezing, leading to a blighting and death of parts or of the entire plant, but it is not so generally understood that less severe frost injury may find expression in the production of permanently dwarfed and crippled individuals. Such injured plants may make a poor growth and

survive for the entire growing season, with little or no fruit production. This behavior is not uncommon in such frost-sensitive plants as beans or cucumbers. The leaves of such plants may be irregular or more or less deformed, chlorosis of parts may be a marked symptom, while blistering or bronzing of the surfaces may cause still further injury.

Annualism in Biennials.—The normal growth of biennials, like carrots, beets, parsnips, etc., during the first season is marked by a vegetative development, the emphasis being placed on food storage in the fleshy root to provide for the production of the inflorescence early the next season. It is a matter of common observation that such biennials sometimes produce their inflorescences during the first season of growth. This phenomenon is very common during some seasons, while in others it is absent or rare. The roots of plants that blossom the first year are of lowered quality, due to a more woody structure and modified chemical composition. This may be illustrated by the sugar beet, the roots of annual plants showing lesser amounts of potash, phosphorus, sulphur and nitrogenous compounds than the roots of normal plants, while there may also be a very material reduction in the sugar content. This is a condition which might be expected, since the inflorescence is developed at the expense of the plastic reserves. This annualism of biennials may be either a character transmitted by the seed or due to the operation of environmental factors.

It may be noted that there is direct experimental evidence that spring frosts during the young stages of growth may furnish the stimulus that initiates the reproductive function. The undercooling of the protoplasm during the spring frosts causes a cessation of growth, and this rest period appears to have the same effect on the nutritive processes as the normal period of winter rest. It is also stated that the later the frost the greater the likelihood that flowering will be induced. It has been shown that subjecting kohl-rabi seedlings to -2 to $-6.5°C$. for 10 hours will induce flowering in 30 to 40 per cent of the undercooled plants. It should not be understood that undercooling is the only cause of annualism, since it is known that other nutritional disturbances may lead to similar results (*e.g.*, length of day in celery).

The Prevention of Frost Damage.—Three general principles used in protection of growing plants from frost are: (1) conserving heat; (2) mixing or stirring the air; and (3) adding heat.

Heat may be conserved by covering the ground or plants with glass, cloth or lath screens, paper caps, etc., or by flooding, as in the case of cranberry marshes. Smudge fires of damp straw or manure are sometimes used to form a protecting blanket over the area. Chemical smoke screens, fogs of moisture particles or chemical-bearing fogs have been suggested (Ext, 1931). Devices for stirring the air with large power-driven fans have not proved practical. This leaves adding heat as the

really important method of protection of orchards or good-sized garden tracts. This may be accomplished by lighting a large number of small fires appropriately spaced throughout the area to be protected. Various types of orchard heaters for use of either oil or solid fuel are available and have been extensively used, with very satisfactory results. By the use of the heaters, the temperature may be raised sufficiently (6 to 10°) to afford safety except in unusual drops. (Young, 1929; Schoonover *et al.*, 1930). Potash fertilizers have been reported to give increased frost resistance to potatoes (Wartenberg, 1929) and also to other truck crops (Wallace, 1926).

WINTER INJURY

Cold Injury to Harvested Crops.—Low temperatures at the end of the growing season or during the period of dormancy or storage may cause injury to root crops or fruit. The principal effects of exposure to low temperature are: (1) The turning sweet of such crops as potatoes, cabbage, etc., when the temperature does not go too low. This gives a desirable flavor to cabbage but an undesirable one to potatoes. (2) The localized killing of tissue or an internal *frost necrosis*, as illustrated by potato, apple and cabbage. The internal black spot of the last appears in some cases to be a frost necrosis, in others a suboxidation phenomenon. (3) Freezing solid, after which the tissues may become soft and watery with the rise in temperature and may suffer ready invasion by bacteria or fungi, or, in some hardy products, there may be a return to normal (see detailed consideration of low-temperature injury of potatoes, p. 171).

Types of Winter Injury to Dormant Plants.—The degree of winter injury to perennial plants, either herbaceous or woody, varies from the most acute effects resulting in death of the plant to localized injury affecting certain organs and evident by either internal or external changes, such as twig blight, dieback, root killing, bud injury, frost cracks, cankers, winter sun scald, crown or collar rot, little leaf and internal necrosis, or blackheart.

Winter killing of winter annuals like wheat, spinach, onions, etc., is frequently responsible for heavy losses. In some localities which normally produce winter wheat, 75 per cent of the fields may be killed out to such a degree as to necessitate reseeding. It is the belief of the writer that winter injury of wheat causes more loss in the Pacific Northwest than the very prevalent bunt or stinking smut, yet but little attention is being directed to this problem. The death of herbaceous perennials during unfavorable winters is of common occurrence. Perhaps the most numerous complaints are of losses to such crops as alfalfa or strawberries. Injured plants may be killed outright, while in other cases, with the advent of spring, there is still a faint spark of life and growth starts, but the vitality has been so weakened that death follows this last struggle for

survival. This is well illustrated in the strawberry, in which the roots and lower portion of the crown are killed while the terminal bud and some adjacent tissue remain alive. In the extreme cases, the buds start into growth, but there are no old roots to function and new roots are not formed; consequently survival is impossible. The exact behavior will depend on the amount of the crown that has survived the winter.

Trees and shrubs, including fruit, shade and forest plantings, frequently suffer severely during tests winters, even in regions to which they are supposed to be adapted. When woody plants are apparently normal at the close of the growing season but fail to start into growth in the spring, the presumption is in favor of death by freezing. A condition in apricot, almond and other stone fruits and more rarely in other than stone fruits, in which the tree dies suddenly as it is coming out in the spring, is characterized by stagnation and fermentation of the sap in bark, cambium and young wood. This so-called "sour-sap" disease is but another type of winter injury.

Twig Blight or Dieback.—Woody plants exhibit two general types of twig growth: (1) a definite or determinate annual growth, in which the twig normally completes its elongation and develops its terminal and other buds by the end of the growing season; (2) indefinite or indeterminate annual growth, in which the twigs make no provision for the winter but continue to elongate until checked by cold. The young, terminal, tender internodes of the latter are invariably killed by the early freezes, so that the new growth comes from lateral buds in the more basal parts. This may be illustrated by the behavior of such plants as the rose, sumac, elder, mulberry, brambles, etc. Dieback is then a normal phenomenon in woody plants having the indefinite annual growth. In many woody plants of the determinate type, twig blight, or dieback, may also occur, since all the twigs are not always sufficiently matured to resist injury by the time of the early winter freezes. In the majority of apple trees, for example, there will be a limited amount of dieback or twig blight almost any season, but this is ignored unless it becomes more abundant and severe. When twig blight is general or when the death of the larger branches takes place, the grower is rightly concerned with the damage, because of its effect upon production and the health of the trees. Species of trees which show a natural tendency to gummosis when tissues are killed by any means will generally show conspicuous gumming of winter-killed branches. This accompaniment of gummosis is very common in the cherry and other stone fruits. Twigs or branches that are killed back or very much lowered in their vitality are easily invaded by semi-parasitic fungi, such as Cytospora, Nectria and similar forms. Even such a parasite as *Sphæropsis malorum* frequently starts its growth in winter-killed twigs or branches and then continues into the living tissues.

Certain species of our fruit trees may show an inherent tendency to grow late into the fall without maturing their wood and consequently will be tender, but in others the failure properly to mature the wood may be induced by excess of soil moisture, overfertility of the soil or various cultural practices. Dieback is not confined to deciduous trees but may cause severe injury in forest or cultivated evergreens.

Bud Injury.—Injury to buds, especially flower buds, may occur independent of dieback or in connection with it. In general, the pistils are the most sensitive of the flower structures and may be killed or injured when other flower parts are uninjured. The winter killing of buds of the peach and other stone fruits is a fairly common phenomenon. Following the injury, the internal tissues will show a brown coloration, and the injured bud will dry up and drop off in the early spring. Winter injury of apple and pear buds is not so common as in stone fruits, but it is probably more common than has been supposed, judging from the studies of Whipple (1912) in Montana. According to this author:

> . . . the important difference between winter-injured apple and pear fruit buds and those of stone fruits is that the former have other possibilities than the production of flowers. In other words, when a fruit bud of the stone fruit opens, it produces only flowers, no leaves or at best only a few rudimentary leaves. It seldom, if ever, produces an axillary growing point. It has no possibilities other than the production of flowers and fruits. If the embryo flower buds within the fruit bud are winter injured, the fruit buds, except possibly in the cherry, seldom open. Such injury could hardly escape notice. In the case of the apple and pear, the buds do open after the flower buds inside them are killed. The bud develops its leaves and, in most instances, an axillary growing point to continue the growth of the spur. The loss of the flower buds might easily be overlooked, and the injured fruit bud would, in such a case, pass for a branch bud.

The degree of winter injury to apple or pear buds is variable, ranging from complete killing of all flower-bud tissue to lesser injuries which lead to the formation of flowers with malformed parts. Some of the effects recorded by Whipple are: flowers as full and double as a rose without stamens or pistils; others with pistils and stamens represented by narrow, ribbon-like petals; pistils absent, other flower parts perfect; pistils and stamens both absent, other parts perfect; and petals absent but other parts normal. The deformed flowers in many cases were noted to produce abnormal fruits, the principal effects being dwarfing, abnormal form and seedless or coreless types. The so-called "tomato" apples afford a striking example of fruit malformation (Abell, 1927).

The winter killing of fruit buds may result when they are in a condition of normal dormancy, provided the temperature drops to a sufficiently low point. Normal dormancy is difficult to define or determine, so it is hardly possible to specify absolute temperatures which will cause the killing of buds in any variety. While killing of buds may be the result of

absolute cold alone, in many environments it is more likely to be due to one or the other of the following: (1) early December freezes while the buds are still immature or not properly hardened; or (2) late freezes following unreasonably warm weather which starts the buds into activity.

Root Killing.—Under certain conditions, trees may suffer from winter killing of the roots. It has been shown that the roots are more tender than the parts above ground and that the small absorbing rootlets are less hardy than the larger roots nearer the crown. Localized or general root killing may result, and, if there is a general dieback of young roots, the absorbing power of the root system may be seriously impaired, with the result that the affected trees make a poor growth. A more general killing of large roots may cause the wilting and death of branches or of the entire tree later in the season after it is in foliage or even after fruit has set. While the young roots are the most tender parts of the root system, they are not injured so frequently as the larger roots near the crown which are more hardy, because the former are generally deeper in the soil and consequently in a more protected position.

It is well known that bare ground will freeze deeper than soil protected by a snow cover or by a cover of vegetation. Root injury is therefore most likely to occur in snowless winters or from extremes of cold when the ground is bare; it is also especially favored by light, sandy soils with poor water-holding capacity.

Frost Cankers.—Localized killing of the bark or of the bark and cambium may result from low winter temperatures. The dead areas are the most frequent on the exposed faces of large limbs, at the forks or crotches of the larger limbs, upon the southwest side of the main trunk (winter sun scald) or at the base of the trunk (crown or collar rot). Bark cankers may vary in size from small, circular or irregular areas on limbs or trunk to extended dead patches which may occupy one side of trunk or branch or completely girdle the bark. The injured area will first show a deviation from the normal in color, and this is followed by sinking and cracking as the dead tissue dries out. Winter-injury cankers may be superficial, involving only outer portions of the bark, while others may be deep, involving the inner bark as well as the cambium. Superficial cankers may cause but little injury, although they may offer an avenue of entrance for certain fungi, but the deep cankers are of more serious character, as they may eventually lead to open wounds which expose the wood and allow the entrance of wood-destroying fungi. Crotch cankers, winter sun-scald cankers, and collar-rot cankers are generally deep seated and seriously menace the life of trees (see special treatment of winter sun scald).

Frost Cracks.—The splitting of trunks or large limbs of forest, shade and fruit trees is not uncommon, but it appears to be least frequent in fruit trees. There are two forms of this type of winter injury: (1)

longitudinal cracks which extend radially from the bark through the sapwood to the center of the tree or beyond; and (2) "cup shake" or cleavage along an annual ring involving a small or an extended part of the circumference. The former is supposed to be due to a rapid contraction of the bark and outer wood, as a result of a sudden drop in temperature, while the warm inner wood does not contract; the latter, or the "cup shake," is supposed to be due to the sudden warming of the outer

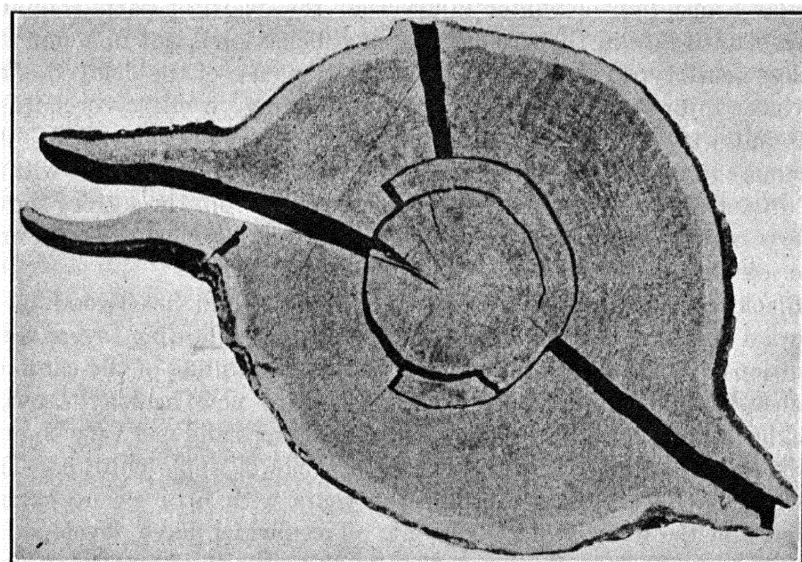


FIG. 57.—Section of tree trunk showing both longitudinal and radial frost cracks. (*After Neger.*)

layers of wood while the inner tissues are still cold. Frost cracks may cause little real harm if the separated tissues come together and heal without the entrance of wood-rotting fungi.

The Little-leaf Condition.—Under certain conditions fruit trees, especially apples, show a trouble which has sometimes been called the "little-leaf disease." This appears to be a type of winter injury that is especially prevalent in orchards in light or gravelly soils with poor water-holding capacity. In this trouble, the tree may start into leaf at the normal time or with only slight delay, but single branches, groups of branches or the entire tree may be so affected that the growth of the leaf clusters is checked before full size is attained, and death results. In the extreme cases, the leaf buds burst the bud scales and expose the clusters of young leaves which may wither and dry up without any further growth. There are all gradations between this condition and those which make a nearly normal development. Wilting and death may be delayed until the leaves are half size or even until later in the season, while in other cases the limb or tree survives but with foliage of reduced size and frequently

of poor color. This progressive death of branches as the season advances often leads orchardists to believe that an infectious disease is spreading throughout the orchard. This type of winter injury appears when there is an absence of any evident lesions on roots, trunk or crown (see also Crown or Collar Rot). These symptoms may result from root injury or when both roots and body tissue suffer derangement or when body vitality has been greatly impaired. It is perhaps contrary to the popular conception of winter injury that the death of parts should be delayed until late in the season, but such behavior is not uncommon in the less acute types of injury. An interesting case of the delayed effects of winter injury was observed in some Washington cherry orchards in 1920 following the severe freezes of early December in 1919. Just previous to maturity of the fruit or even later, the foliage would wilt and dry up and the fruit shrivel. Much of the fruit from these trees showed a very poor keeping quality in transportation to market, apparently suffering because of the weakened vitality of the trees.

Blackheart or Internal Necrosis.—The pith and heartwood or the sapwood also may show a pronounced blackening following severe winter conditions. This change may occur without the killing of the cambium, which will continue its activity and produce new healthy sapwood. Blackheart may occur in either young or old trees and is a very common condition in apple trees in the northern range of apple culture. Trees showing blackheart may continue for years with little or no external evidence of the internal derangement. Trees which have developed the internal necrosis are very susceptible to the inroads of wood-destroying fungi, the necrotic tissue being much less resistant to the advance of certain fungi than normal wood. Not all cases of blackheart are due to winter injury, since it appears to be a characteristic feature of silver leaf when healthy trees are invaded by *Stereum purpureum*. It appears that essentially the same changes in the tissues may result from either fungous invasion or freezing. A silvering of the foliage appears to be one of the aftereffects in both cases. Silvering of the foliage as an aftereffect of winter injury has been noted in the apple. A pronounced case was noted in the prune orchards of the Walla Walla, Wash., district following the severe winter of 1919 and 1920.

Factors Affecting Winter Injury.—The degree and the type of winter injury will be influenced by the condition of the plant as well as by the combination of unfavorable weather conditions. It is not alone the absolute temperature or the minimum cold which is of importance but the time in the period of dormancy when the cold is experienced, and whether gradual or sudden changes occur. Periods of zero weather in December are very likely to cause severe injury, because the tissues have not yet become hardened by the longer action of moderate cold. Periods of zero weather following moderate weather which starts tissues into activity

are also likely to induce heavy injury. The presence or the absence of a snow cover at the time of the heavy freezes is also a matter of much importance.

The type and the severity of winter injury will be influenced by the following factors: (1) the species or kind of crop and the variety within the species; (2) the age in the case of perennial plants; (3) the degree of dormancy of the plant, as a whole, or of special parts at the time of critical temperatures. Some of the factors which affect not only dormancy but other features which influence hardiness are: (1) kind or degree of pruning and time of pruning; (2) the amount of crop produced the previous season; (3) the heat and light income during the growing season, especially during the late fall; (4) the physical characters of the soil and subsoil; (5) the natural fertility of the soil or the fertilizing practices; (6) the site with reference to soil moisture or drainage conditions; (7) the time of irrigation and the amount of water used; (8) the presence or absence of cover crops; (9) cultural practices, *e.g.*, time of seeding for cereals or furrow seeding (Salmon, 1924).

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LOW-TEMPERATURE INJURY OF POTATOES

Potato shoots are especially frost sensitive and are frequently killed by late spring or early fall frosts. A frost necrosis of leaves in the nature of minute brown spots on leaves otherwise normal has been attributed to low temperatures (Macmillan, 1920). Growers, dealers and consumers are probably more concerned with the effects of low temperatures upon the tubers, either at maturity in the field, during late harvesting or during storage or transit to market.

Symptoms and Effects.—Three prominent types of injury to potato tubers may result from the action of low temperatures:

1. *Freezing Solid.*—Tubers held at a temperature at or below the freezing point for potato tissue freeze solid—either the entire tuber or on one side or end. Tubers close to the surface of the ground or slightly protruding are sometimes caught by the sudden early freezes. When frozen tissues are killed and then thaw out, decompositions set in, the tissue becomes softened, the skin is raised and ruptured by gas accumulations and a watery exudate oozes out. The cells beneath the skin are loosened by the solution of the middle lamellæ, and a cut surface immediately turns brown. In case of partial freezing, a dark line may separate the frozen tissue from the normal. In many cases, decay by either bacteria or fungi will set in, and the partially frozen tubers will be still more injured; while under other conditions, the frozen tissues may dry out and shrink without decay and later may appear dry, whiter and more powdery than normal.

2. *Turning Sweet.*—Tubers which have been stored for a number of weeks at temperatures closely approaching the freezing point for potato tissue develop a sweetish taste. This "turning sweet" has been thought by some to be due to slight freezing and has been popularly termed "chilling." While this sweetish taste may be objectionable, it causes no lasting injury, since the tubers will again become normal with exposure to higher temperatures.

3. *Internal Frost Necrosis.*—Tubers which have been subjected to low temperatures but not sufficiently low or sufficiently long to freeze solid may develop internal discolorations or necrotic areas which are evident

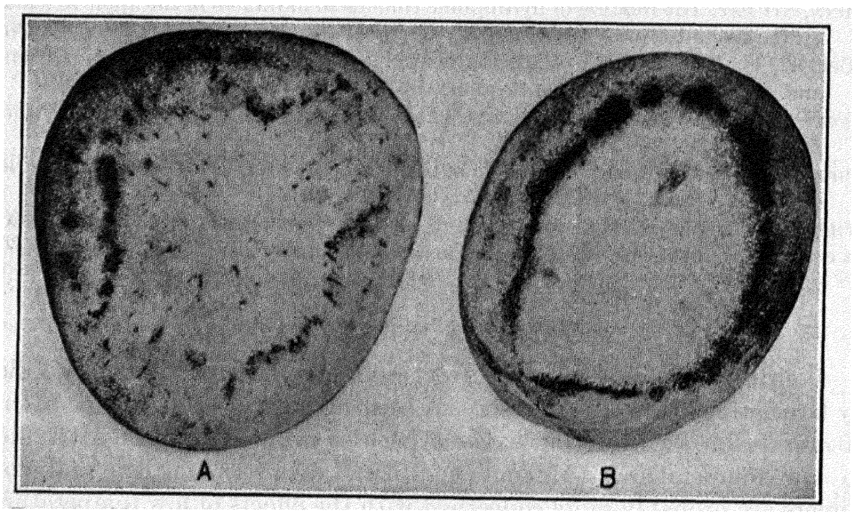

FIG. 58.—Net and ring types of frost necrosis of potato. *A*, intense net discolorations in both medulla and cortex; *B*, intense ring type somewhat complicated by blotch. (*After Jones, Miller and Bailey, Wis. Res. Bul.* **46**.)

upon cutting into the flesh. Three different types of discolorations have been recognized as due to low temperatures: (1) the *blotch type*, appearing as ovoidal or irregular patches ranging from a slight metallic tinge to opaque gray, dark brown or almost sooty black and located most frequently in the cortex or in the vascular ring, although sometimes present in the pith; (2) the *ring type*, characterized by lesions in, or adjacent to, the vascular ring, making a continuous or broken ring, narrow and distinct or broader and more diffuse, and showing the same shades of color as in the blotch type; and (3) the *net type*, evident as a browning or blackening of the fine ramifications of the vascular elements, so arranged as to give a broken, net-like pattern, either exterior to, or within, the vascular ring. The internal necroses are most evident toward the stem end of the tuber and in slight injuries may be restricted entirely to that part. They are not indicated by any external markings, and the injury is generally evident only when the affected tubers are cut in two.

In severe internal necrosis, shriveling may be increased, internal splits or pits may form, while Fusarium dry rot may enter. If dry rot proceeds to its later stages, the distinguishing symptoms of frost necrosis are soon obliterated (Jones *et al.*, 1919). Blackheart (p. 132) and other internal lesions may complicate those of frost necrosis. Special mention should be made of the possible confusion of the net type with the heritable net necrosis of leaf roll and of similarities of the ring type to the bundle browning which frequently accompanies the wilt diseases. In the case of

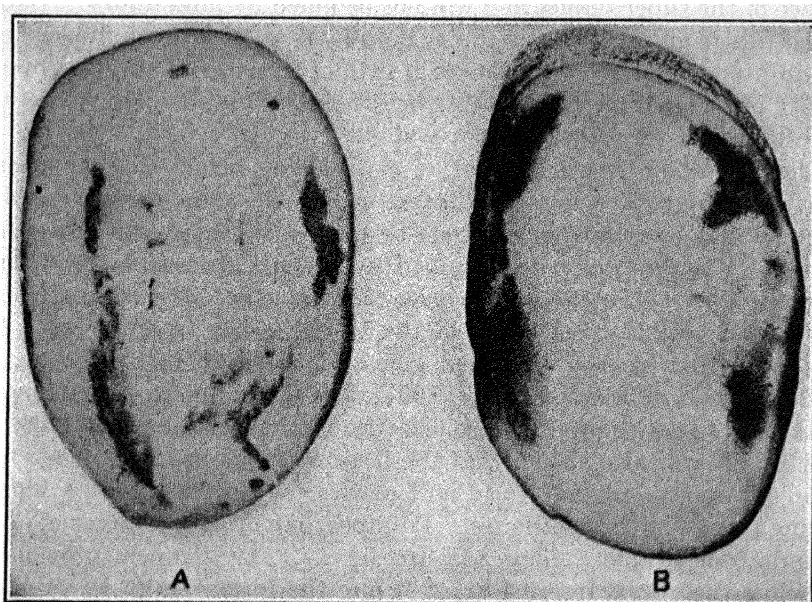

FIG. 59.—Blotch type of frost necrosis of potato. *A*, longitudinal section; *B*, cross-section showing intense vascular and cortical blotches that were evident on the exterior as dark areas. (*After Jones, Miller and Bailey, Wis. Res. Bul.* **46**.)

frost necrosis, some of the tubers will show one type of lesion, while other types will be found in other tubers of the same lot; hence the examination of a considerable number of tubers should make a diagnosis fairly certain.

In addition to the three types of injury already described, low temperatures may be either directly or indirectly responsible for injury. If immature potatoes which have suffered skin injury in harvesting are stored at once at temperatures below 40°F., shriveling and some breakdown may follow, since wound cork will not be formed. Storing at 32 to 36°F. very soon after digging may also cause darkening and killing of buds or the weakening of buds with formation of spindling sprouts when growing conditions are furnished, while in other cases there may be a darkening and necrosis around the lenticels (Peacock and Wright, 1927). "Contact" frost injury may result from local freezing solid or local action of temperatures sufficient to cause necrosis, from contact with ice, frozen-

solid tubers, ironwork in cars or from other objects that are very cold (Tucker, 1928). A killing of tops by a severe frost before maturity may be responsible for darkened, thread-like areas which radiate from the stem end.

The injury to tubers intended for table use depends on the extent of the internal necroses and the degree to which rot-producing organisms become established. Frost-injured tubers are not reliable for seed purposes, although the eyes are more resistant to freezing injury than the balance of the tuber tissues and will not be killed by mild injury. Badly frosted tubers give an unsatisfactory germination, while even moderately frost-injured tubers show (1) slower growth of the sprouts and (2) greater liability of the seed pieces to rotting before the young plants have become well established on their own root system.

Etiology.—The freezing point of potatoes is below that of water, since their sap is a solution of salts, sugar and various other soluble materials. It has been shown that the expressed sap freezes at a point closer to 32°F. than the potato tissue itself (30.8°F. according to Müller-Thurgau, 1880). The actual freezing point of potatoes varies with the variety and with the condition of the tubers. The freezing point for potatoes during storage has been given as 28 to 26°F. by Appleman (1912), while Wright and Harvey (1921) determined the freezing point of 18 different varieties by the thermoelectric method to vary from 29.66 to 28.13°F. It was also shown that the freezing point tends to rise as the season advances and that early and midseason varieties have a higher freezing point than late varieties. Potatoes, like other plant tissue, must be undercooled before they will freeze; *i.e.*, the temperatures must fall below the actual freezing point before the formation of ice crystals begins. The temperature then rapidly rises to the freezing point. The undercooling range is variable and depends on the variety, the rapidity of the temperature fall and the air temperature. This may be illustrated by two cases, one showing undercooling to 20.2°F. and freezing at 29.15°F., while the other undercooled to 23°F. and froze at 29.15°F.

> As undercooling advances, the stability of the liquid state of the sap within the tissue becomes more and more strained until a point is reached when the slightest perceptible jar will terminate the undercooling and freezing will begin instantly (Wright and Harvey, 1921).

But undercooling may be terminated at any temperature below the freezing point.

The turning sweet of potatoes may be noticeable at 35°F., but it has been shown to take place most rapidly at the freezing point of water or below (Appleman, 1912), when the temperature does not drop sufficiently low to cause freezing solid. This change is due to the gradual transformation of starch into sugar which, with the retarding of the life processes,

accumulates in the cell sap. This increased concentration of the cell sap may have a protective value by lowering the freezing point. When potatoes which have turned sweet are again exposed to higher temperatures (10°C. or more), respiration becomes more active and the sugar is consumed. Sudden lowering of the temperature will not cause potatoes to become sweet, but they must be exposed to unfavorable temperatures for a fairly extended period, as the sugar accumulation is a slow process.

The various types and patterns of internal discolorations which are characteristic of frost necrosis in potatoes under storage conditions have been produced experimentally. According to Jones *et al.* (1919):

> In general, frost necrosis will appear in at least a portion of tubers which are subjected to a temperature of −10°C. for 1 hour, to −5°C. for 2 hours or to −3°C. or slightly lower temperatures for several hours.

This conclusion was based on tests made with the Rural New Yorker. Wright and Taylor (1921) using seven different varieties at 28°F. found very little internal necrosis with exposures up to 48 hours, but, with exposure for 70 hours, 31 per cent showed injury. When held at 25°F. for 5 hours, there was no injury; but for 19 hours, 16⅔ per cent were injured; for 24 hours, 46.7 per cent; while exposure for 43 hours injured over 52 per cent, a few being frozen solid.

Frost necrosis is due to localized formation of ice crystals with the resulting death of the cells.

> Since the lesions of frost necrosis result directly from the oxidation of cells killed during the freezing process, they are not evident in tubers when they are first removed from the freezing chamber but appear only after such tubers have been exposed to warm air for several hours (Jones *et al.*, 1919).

According to these authors, the color changes which occur during the oxidation process

> . . . range through pinks, browns and grays and seem to develop simultaneously throughout the injured tissues. The time required for the ultimate dark color to be reached depends in part upon the air temperature; thus, at temperatures of 10 to 15°C., from 10 to 12 hours were required; while at 25 to 30°C., only 5 or 6 hours were necessary. There was no evidence that the rate of thawing influenced the degree of injury nor that tissues which had received severe freezing injuries blackened more rapidly than did those with lesser injuries.

The act of terminating the undercooling process after the freezing point has been passed, by some mechanical means, such as jarring or tapping, has been termed "inoculation." The closer the temperature is to the actual temperature necessary for crystal formation the more easily does inoculation take place. "When undercooled, jarring resulting from rough handling or incidental to hauling is liable to cause potatoes to freeze" (Wright and Taylor, 1921). This effect of jarring has been shown by experimental tests in which undercooled potatoes were dropped

certain distances. When undercooled to 28°F. and dropped 2 feet, potatoes bruised badly and showed severe freezing injury; but when dropped 1 foot, they did not bruise but showed freezing injury; while when dropped once 6, 4 and 2 inches, no injury resulted; but when dropped six times, frost injury followed. When undercooled to 25°F., potatoes are more sensitive to jarring than they are at 28°F. The danger of handling potatoes when undercooled is illustrated by the severe freezing injury which resulted when sacks of potatoes held at 28°F. were rolled across the floor a distance of 50 feet.

Prevention of Freezing Injury.—Tuber injury may result from low temperatures before digging or during storage or transit to market. The following preventive features may be emphasized: (1) Dig the crop before severe frosts. (2) Maintain the temperature of the storage room as nearly as possible between 35 to 40°F. This will prevent the potatoes from turning sweet and also will prevent internal necrosis. If potatoes are not handled, little or no injury will result if the temperature drops for some time to 28°F., but potatoes held at 32°F. have given poorer results for seed (Wollney, 1889) than those stored at higher temperatures. (3) Potatoes shipped during cold weather should be provided with artificial heat and should be so loaded as to provide for a free circulation of the air to all portions of the car. (4) Avoid the handling of undercooled potatoes until the temperature has risen above the freezing point. (5) The types of freezing injury which result from immediate storage at cold-storage temperature may be avoided by holding in preliminary storage at higher temperatures. This protection has been afforded by 3 weeks at 40 to 50°F. or 7 days at 60 to 70°, after which storage at 32° was safe. In cases of heavy freezing injury, it has been shown that the affected potatoes are still of value for the production of starch, as freezing does not affect the starch grains (Edson, 1918).

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FREEZING INJURY TO FRUIT

Freezing injury to mature fruit may result before it has been removed from the field to the packing plant, in common or cold storage or during transit to market.

Freezing injury does not always occur when fruit or vegetable products are exposed to temperatures at or below their actual freezing points. Under certain conditions, many of these products can be undercooled, *i.e.*, cooled to a point below the true freezing temperature of each and again warmed up without freezing and without apparent injury. Certain products under certain conditions may be actually frozen and then thawed out without apparent injury, while, on the other hand, some products are injured if stored at temperatures well above their actual freezing points (Wright and Taylor, 1923).

Apples.—The freezing point of apples has been shown to vary from 26.87 to 30.16°F. on the basis of a large number of determinations on 10 varieties (Carrick, 1924). The average freezing point for a large number of varieties from eastern and western orchards has been placed at 28.5°F. (Diehl and Wright, 1924). Undercooling may sometimes be as much as 7 to 8° below the freezing point without causing ice formation if the apples are not disturbed. Low-temperature injury to apples may be invisible or visible. Some of the effects of freezing are (1) changes in composition, flavor or texture; (2) increased injury from bruising; (3) increase of susceptibility to decay-producing organisms; (4) premature physiological breakdown, possibly as a result of increased

respiratory processes; (5) discolorations, both internal and external, in moderate injury involving only vascular necrosis or skin discolorations; (6) moderate or severe disorganization of tissue resembling maturity breakdown.

A type of freezing injury different from anything known in apples has recently been described for pears (Hartman, 1931). The injured pears have a glassy, water-logged appearance, the water-logged tissue being located in the outer portion of the fruit or sometimes also within the core, while the intervening portion is dry and pithy. This injury is reported to be caused by long-continued freezing (4 to 6 weeks) at temperatures ranging from 23 to 27°F.

Tomatoes.—Many cars of tomatoes are shipped each year from southern points during the freezing temperatures of winter and early spring months. The average freezing point of 19 commercial varieties was found to be 30.46°F. (Harvey and Wright, 1922). Considerable undercooling (to 22.63°F.) may take place before actual freezing. The first visible sign of freezing to death of fruit tissue is the appearance of small or extensive water-soaked spots or areas. Fruits do not freeze so readily as the foliage, and those in contact with the ground may be less injured than those free from the soil. Temperatures which have not been sufficiently low to cause immediate injury may be detrimental. Tomatoes held for 4 days at 32°F. showed no injury, and ripening proceeded normally when exposed to room temperatures, but longer exposures (8 days) lead to breakdown and decay (Diehl, 1924). A yellow blotching of fruit several days following a severe frost has been noted, and apparently uninjured fruits picked following a frost have been reported to decay more than normal stock (Wright *et al.*, 1931). Chilling of green tomatoes (even at 25°F. for 18 to 21 hours; 5 to 8 days at 32°; or 11 to 15 days at 40°F.) does not prevent normal ripening when removed later to higher temperatures, but ripening may be delayed.

Grapes.—The range of freezing points (Carrick, 1930) has been given as follows: Emperor, 23.63 to 28.66°F.; Flame Tokay, 23.85 to 27.66°F.; Malaga, 24.47 to 28.66°F. The average undercooling for the three varieties was 5.22°F. The effects following freezing and subsequent thawing are: (1) *Color changes:* the Emperor is darkened and is more translucent and water-soaked toward the stem end; the Malaga becomes darker green and, with severe freezing, a distinct brown sometimes as deep as russet. (2) *Wilting and viscosity of skin:* slight wilting to pronounced wrinkling, the degree varying with the severity of the freezing, is a marked feature. The viscosity of the surface is due to the exudation of sugar. (3) *Texture and flavor changes:* crispness and turgidity are reduced, and with severe freezing the berries become watery and insipid. (4) *Susceptibility to decay:* increased susceptibility to Botrytis infection is an after effect of freezing.

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CROWN ROT OF TREES

The term "crown rot" or "collar rot" is used to designate a bark disease in which the primary lesions are located in the bark of the basal part of the tree trunk or on adjacent portions of the large roots. Splitting and death of bark are followed by decay, and the affected tree may be partially or completely girdled, resulting in serious derangement of functions or in death.

Symptoms and Effects.—The most striking symptoms of crown rot of fruit trees can be noted during the growing season following the dormant season in which the initial injury occurred. Following are the noticeable features: (1) a scant foliage with leaves of small size; (2) pallor or chlorosis of the foliage; and (3) a sickly coloration of the bark of trunk and limbs, especially in smooth-barked species. In the apple, for example, the bark assumes a reddish-yellow cast which makes it possible easily to pick out the affected trees from a distance. A portion of the tree or the entire tree may show the symptoms of the disease.

The symptoms described are practically certain indications of the presence of well-developed lesions at the base of the trunk or on the larger roots, where bark will be found to be dead and discolored, either on one side of the trunk or completely encircling it. The bark soon becomes sunken, cracks or fissures may appear and during the progress of the season the dead bark disintegrates and weathers away somewhat and finally the underlying wood may be more or less exposed. The injured bark may be largely at the ground line and below, at the junction of large roots with the trunk, or it may be at the ground line and extend 12 to 18

inches or more up the trunk. Especially in portions of lesions protected by the soil, the outer portion of the bark may be left intact while the inner portions may disintegrate into a granular mass which becomes powdery when dry. The wood beneath affected bark may become brownish or even charred in appearance. Lesions may extend gradually, advancing downward most rapidly, less rapidly upward and rather slowly laterally. If bark disintegration is checked, callus may be formed, and more or less healing of the wound may follow. In some cases, all of the bark is not killed down to the cambium, and under favorable conditions regeneration of tissue may occur in sufficient amount to prevent the development of an open canker.

The final result of crown rot will depend on the depth and surface extent of the lesions and probably also on the soil and moisture conditions which prevail during the season following the initial injury, while the abundance of wood-destroying fungi in the environment may play a part. In the severe type in which the bark is killed down to and including the cambium, serious results are likely to follow. Complete girdling will, of course, prove fatal unless bridge grafting is resorted to, and cankers involving three-fourths of the circumference of the trunk will cause serious interference with the life of the tree. Cankers involving only part of the circumference may be extended the next season and cause complete girdling, or the advance may be checked.

Etiology.—A survey of the literature concerning collar rot shows that troubles having very much the same symptomology have been attributed to a variety of causes. Most of the evidence points to the fact that the disease in the majority of cases is but one phase of winter injury somewhat akin to winter sun scald and crotch cankers. Collar rot or root rot may, however, occur independent of winter injury, sometimes being due to bacterial invasion (see Fire Blight) or various fungous parasites (see Mushroom Root Rot). Headden (1908–1910) in Colorado has attributed the trouble to arsenical poisoning, while alkaline irrigation water has been looked upon as an inducing factor or as contributing to the prevention of healing after the lesions have been formed by other agents (Hotson, 1920). There is no doubt that wood-rotting fungi play a secondary part by invading the wounds, whatever their primary cause may be.

The conclusion that most cases of crown rot of fruit trees are due primarily to low temperatures and excessive or late fall growth is based largely on field observations (Grossenbacher, 1912; Thomas, 1926; Magness, 1929) rather than on experimental evidence. It has been very definitely shown that the early stages of the crown rot originate during the winter or dormant season. The first visible stages "consist of discolored and often ruptured tissues variously distributed in streaks and patches in the bark," and this condition can be found in the late winter and spring. The progress of the trouble following the initial injuries is

rather varied, and the tissue changes are somewhat complex. The initial injuries may be outgrown, or typical cankers of severe type may develop. The non-parasitic character of the disease is supported by the fact that no organisms of any kind are commonly found in young lesions. The base of the tree trunk matures its tissue more slowly than portions higher up and consequently is susceptible to injury from freezing in the late fall or early winter. Crown rot is, therefore, believed to be an early form of winter injury, in contrast to sun scald, which is a late winter or spring form of injury.

Prevention and Treatment.—There is no possibility that crown rot can be entirely prevented, but attention to cultural and irrigation practices will give some relief. Effort should be made to keep trees from growing too rapidly or too late into the fall in order that they may not be subjected to unfavorable temperature when the bark is still immature. It has also been suggested that varieties of trees which are subject to this type of winter injury should be headed low regardless of the inconveniences which may be experienced in cultivating. The swaying of young trees by the wind is believed to play some part in the production of the initial injuries; hence windbreaks might prove of special value in lessening the disease.

In orchards in which crown rot prevails or when conditions have been such as to indicate the probable occurrence of the disease, the trees should be examined during the spring and early summer for indications of bark injury at the base of the tree trunk. In case such injuries are found, the soil should be removed from contact with the injured portions, and all of the dead bark cut out. The cut surfaces should then be treated with an antiseptic and waterproofed, or a combined antiseptic and waterproofing treatment may be substituted. Waterproofing alone, using coal tar or grafting wax, would be sufficient if one could be certain that no organisms were concerned. In cases of complete girdling or when the lesions involve a large part of the circumference, trees may be saved by resorting to bridge grafting or perhaps in other cases preferably by approach grafting (Thomas, 1926). It has been shown by more recent work that uncovering the injured crown not only offers better conditions for healing following injury but increases the resistance of the exposed parts to freezing injury (Magness, 1929). This gives support to the idea that injury might be avoided by guarding against too deep setting of trees when planting the orchard. Moderate pruning and removal of fruit are also believed to facilitate recovery.

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WINTER SUN SCALD OF TREES

Bark injury on any portion of a tree may result from the freezing to death of the tissues, including the primary cambium and the external parts, or in superficial cankers the primary cambium and inner bark cells may escape injury. When the bark injury is localized, more or less

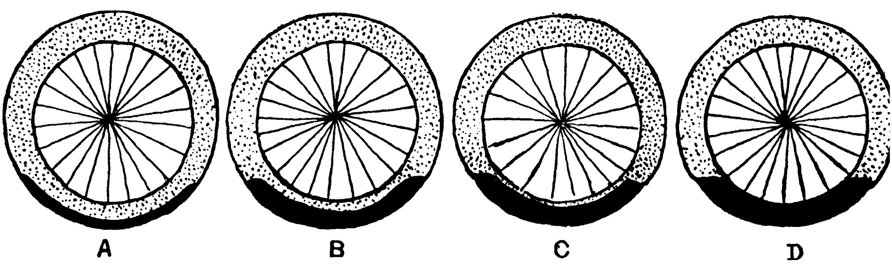

FIG. 60.—Diagrammatic cross-sections of tree trunks showing superficial, moderately deep and deep sun-scald cankers.

well-defined dead areas are formed, which with cracking and peeling of the dead bark may become open wounds or cankers. These cankers most frequently occur on the southwest face of the tree trunk, from the ground up, on the sun-exposed surfaces of large branches or at the junction of scaffold branches with the main trunk. Whenever the freezing injury is localized on sun-exposed surfaces, it is classed as "winter sun scald," a term used to distinguish the trouble from summer sun scald due to the killing effect of high summer temperatures. Sun scald of some thin-barked trees from overheating during the intense sunshine and high temperatures of midsummer, coupled with drying out of the tissues, does undoubtedly occur, but winter injury is probably a much more common cause of cankers, on both fruit and forest trees.

Symptoms and Effects.—The early stages of winter sun scald are frequently not observed. Following the period when the injury occurred, the affected bark may appear dull or discolored, and there may be some shrinkage due to drying out of the tissues. Soon the bark will be found to be loose from the wood, and the brown, dead portions can be readily peeled away from the underlying wood, which may also be discolored.

In the more severe types of injury, the bark may split and crack quite soon, or this may be somewhat delayed, but the final result in the undisturbed cases will be the weathering and peeling of the bark to produce more or less of an open wound. In the more moderate types of winter sun scald the injury may be confined to a narrow strip on the southwest face of the trunk, while in more severe cases the bark may be killed for nearly the entire circumference of the tree. The immediate injury will depend on the extent to which girdling has been completed and the func-

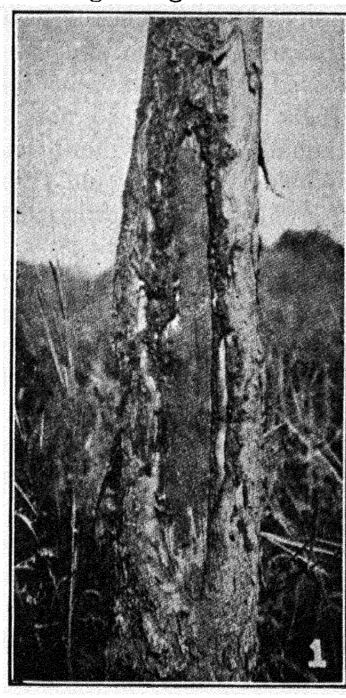

FIG. 61.—Old winter sun-scald canker on apple. (*After Mix, Cornell Bul.* **382**.)

tional activity of the remaining portions lowered. The most severely sun-scalded trees may die later in the season; while in those less severely injured, healing will take place and the trees may survive if the entrance of wood-rotting fungi can be prevented. This secondary injury from the entrance of wood-rotting fungi is a very fruitful cause of the final decline of trees. The disintegration of the wood by fungi which enter through sun-scald cankers or crotch cankers may so weaken a tree that it goes down during the stress of wind storms.

Etiology.—It was at one time believed that winter sun scald was due to the rapid thawing of the frozen tissue on the sun-exposed face of the trunk or large branches. The present opinion is that the injury is the result of the direct freezing to death of the tissue. This conceivably might result from one or the other of two ways or by a combination of these:

1. An increased tenderness in the tissue on the sunny side of the trunk in late winter causes it to kill at a temperature not low enough to injure the still dormant tissue of the shaded side.

2. The tissue on the sunny side kills at a higher temperature, due to the more rapid temperature fall that may take place after a cold, sunny day in later winter (Mix, 1916).

As a result of freezing tests carried out by Mix, the conclusion was reached that apple bark on the southwest and on the northwest sides of the trunk showed no appreciable difference in hardiness and that therefore the view that the warmth of the sun, by promoting metabolic activities, caused increased tenderness must be eliminated as the cause of sun scald. It is of interest to note, however, that trunk tissue from all sides gradually becomes more tender as the end of the dormant season is approached. This would suggest that sun scald is more likely to occur in late winter or spring than in the early part of the dormant period.

The view that freezing to death is due to a "rapid temperature fall consequent to a warming up of the tissue above freezing by the rays of the sun on a bright, cold day in late winter" is supported by observations and experiments: (1) the higher day temperature of the southwest side of tree trunks as contrasted with the northeast side varying from a slight excess to a maximum of 20° or more; (2) the equalization of temperatures on both sides during the night periods; (3) the increased killing of buds or twigs known to result from rapid freezing as contrasted with slow freezing. On the basis of these facts, winter sun scald is supposed to be a late-winter injury. It is not the result of increased sensitiveness due to late growth or failure of tissues to mature but is likely to occur any season whenever the rate of temperature fall on the sun-exposed side is sufficiently rapid following the warming up by the winter sun, and the minimum reached is sufficiently low.

Prevention.—In many regions, winter sun scald may be of such rare occurrence that preventive measures are not justified. In those regions where sun scald is frequent, practices may be followed which will prevent the excessive heating of the sun-exposed bark. This may be accomplished in either of two ways: (1) by shading to protect the bark from the direct action of the sun; or (2) by modifying the absorption of heat with full exposure. The use of board or lath screens has been recommended with trees that are headed high, while very low heading of trees has been practiced as a protective measure with some success.

Spraying or painting the trunk and large branches with whitewash has been quite generally recommended for northern regions where winter sun scald is common. The protective effect is due to the fact that the whitewashed bark does not heat up to such high temperatures as normal bark during the warm winter days. In the treatment of sun-scald cankers

by the removal of all dead and decayed bark, the recognized methods of disinfection and protection of the wounds should be practiced.

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CHAPTER IX

DISEASES DUE TO UNFAVORABLE LIGHT RELATIONS

Before considering the ways in which disturbances of the light relation may bring about derangements in the life of our cultivated plants, brief record should be made of the part which light plays in some of the important physiological processes.

The Function of Light in the Life of the Plant.—Light is essential (with few exceptions) to the formation of the green pigment, chlorophyll, and furnishes the power or energy by which the chlorophyll apparatus is able to use the crude materials, carbon dioxide and water, in photosynthesis, or the construction of carbohydrate food. The output of our plant factories depends upon the intensity, duration and quality of light; hence it must be evident that the amount of plastic material available in plants for assimilative and growth processes bears a definite relation to illumination.

That light has a direct effect upon living protoplasm is evidenced by various heliotropic curvatures or movements of plant organs, which direct them either toward or away from the light, by heliotactic movements of motile plant protoplasts or by intracellular changes with variations in the intensity of light. Since protoplasm exhibits a marked sensitiveness to light, it is only reasonable to expect that growth will be affected by light conditions. Growth is favored by diminished light intensity and retarded by bright illumination. During the daylight period, the energy of the plant is directed to the work of food manufacture; while during the night period, photosynthesis ceases, and the reserve of plastic materials which was accumulated is available for constructive work. Ordinary daylight does not cause a cessation of growth but merely a retardation. This behavior is illustrated by the growth increments of certain organs which in darkness may be more than double those for similar periods of ordinary daylight. While light is not essential to the germination of most seeds, it is the stimulus that in some cases unlocks the chemical changes that initiate germination.

Transpiration is a third physiological process affected by light. The effect may be either direct upon the living substance or upon the cell machinery or indirect by modifications of the plant structures. Since transpiration is not comparable to simple evaporation from an exposed water surface but is a process controlled more or less by the chemical and physical properties of the protoplasmic body from which the moisture must be withdrawn, light may affect transpiration by modifying the

permeability of the protoplasm and thus accelerate or retard the process. By affecting the osmotic pressure in the guard cells or adjacent epidermal cells, light may cause an opening or closing of the stomata and thus promote or retard *stomatal transpiration,* while cuticular transpiration will be influenced by the thickness of the epidermal walls, their cutinization, the character of the cuticle and other structural features which are modified by light intensity.

Shade Plants and Sun Plants.—Brief reference may be made to the fact that some plants in their natural habitats may be able to make their best growth only when exposed to the full intensity of normal light, while others thrive best in partially shaded localities in which the light is of moderate intensity. Thus we may have sun plants at one extreme and shade-loving plants at the other, while others are less sensitive to their light environment. These natural peculiarities must be taken into consideration in providing favorable conditions for plants under cultivation. Shade-loving plants may suffer injury if exposed to the full intensity of the sun, or sun plants may make a poor growth if planted in poorly lighted environments. Because of the injurious effects of intense light on the seedlings of coniferous and some other forest trees, lath screens are used in forest-tree nurseries to give approximately half light.

General Effect of Light Deficiency.—For a normal thrifty development, a certain intensity and duration of light are essential. It is difficult to fix upon any definite optimum, but it must be recognized that when the light income of a plant sinks below the minimum requirement or rises above a maximum, the plant will cease to thrive. The *optimal light income* denotes the amount of light (measured by intensity and duration) which will induce the best growth or produce a type of development that is most nearly normal. As the light income drops from the minimum to zero or complete darkness, the plant may undergo gradual, formative or structural changes, including alteration of color and peculiarities of structure which are characteristic of the condition known as etiolation. The sickly yellow plant that has been entirely deprived of light represents the extreme of etiolation, while, with exposure to light, the symptoms become less and less evident until normality is attained.

The changes which are characteristic of etiolation are as follows: (1) the *abnormal elongation* of stems (internodes) and petioles or of leaves that are normally elongated and have a basal or intercalary meristem, thus producing shoots that may be characterized as "spindling." This modification occurs in plants with stems that are normally elongated and also in those of the rosette habit. (2) The pronounced reduction in the size of the leaves (shade leaves in many cases are larger than sun leaves). This feature is characteristic of dicotyledons, while monocotyledons generally show an elongation and narrowing of the leaves, although deviations from this behavior may be found in both groups. (3) The

reduction in the amount of chlorophyll or its complete disappearance (in darkness) and consequently the slowing up of photosynthesis or a complete cessation of food manufacture. In complete etiolation, growth can continue only at the expense of food reserves of seeds, modified stems or other storage organs. It should be noted here that etiolation is not the result of a checked or inhibited photosynthesis, since plants will not make a normal development in darkness even when supplied with an abundant reserve of plastic food. (4) The suppression of the reproductive function, as illustrated by sterility, lessened flower formation or the complete absence of blossoms as a result of shortage of food. Plants like hyacinths, tulips and narcissuses, with an abundant food reserve, will develop nearly normal blossoms in complete darkness. (5) A soft or succulent type of growth. While the stems are more slender and leaves thinner, cell walls are more delicate, and there is a poorer development of mechanical tissue. The general effect of diminished light is to cause a poor development of the palisade parenchyma of leaves, so that shade leaves may not be more than half as thick as those developed under normal illumination.

Plants grown under poor light conditions wilt more readily than normal plants when exposed to bright light. This may be illustrated by the behavior of cucumbers grown under glass.

Plants grown under the poor light common to November and December have leaves of poor color, slender and elongated petioles and little mechanical or resistant tissue, and when subjected to the bright sun in the early spring every plant in the house will wilt (Stone, 1913).

Poor light, or "partial etiolation," renders plants more susceptible to the attacks of fungous diseases. Undoubtedly the host modifications play an important part in this increased susceptibility, while at the same time the reduced light intensity offers conditions more favorable to the growth of fungi. The light factor is not the sole favoring condition, for diminished intensity of light is generally accompanied by increased humidity of air, which may also affect both host and parasite. As an illustration, it may be noted that lettuce grown under glass sometimes suffers severely from leaf blight and stem rot, due to Botrytis, during the dark, cloudy days of winter, while the trouble may largely disappear with the advent of days of continuous sunshine. Lack of light together with excessive moisture increases the susceptibility of foliage to injury from fumigants—*e.g.*, burning from hydrocyanic acid gas—while insufficient light income has an important bearing upon one type of spray injury (see Lime-sulphur Injury, p. 230). Insufficient light produces a type of growth in which maturity is delayed, and consequently winter injury is more likely. It seems that winter injury in some environments is accentuated not alone by the moderate temperatures of the fall preceding

freezing weather but also by the lowered light income due to cloudy, foggy weather.

Etiolation in Horticultural Practice.—While etiolation represents a derangement of normal physiological processes, it may be utilized to produce desirable modifications of behavior or to produce structural modifications which render the plant more desirable as a commercial article. Hyacinth bulbs planted in flower pots in the fall sometimes show a delayed development of the leaves and the flower stalk, which remain short. This may be prevented, in part at least, by covering the bulb and bud with a cap of dark paper, which will exclude the light. This induces etiolation, and, as a result, the leaves and flower stalk elongate. Similar use may be made of withdrawal of light to induce the formation of blossom shoots in other plants.

Etiolation or blanching gives a desirable quality sought in certain vegetables—*e.g.*, asparagus, endive, celery, French globe artichoke, head lettuce and cabbage. The etiolation may be induced by depriving the parts to be blanched of light; but in numerous cases, cultivation and selection have produced varieties that are partially self-blanching, the failure to green becoming a hereditary character. Etiolation improves or modifies the flavor and produces crisp, juicy and tender tissues, a desirable feature, especially in salad plants.

General Effect of Intense Light.—If plants are in danger of sunstroke, they are not able to flee to a shaded or secluded spot where the light is less intense, but they do, by certain habits or by active responses, show a sensitiveness to the intense light of the environment in which they live, and these may be purposeful responses which serve as a protection against injury from intense light. A number of illustrations may be noted. The chlorophyll apparatus of the leaf is in the most favorable position for receiving the full intensity of the light when the rays fall most nearly perpendicular to the leaf surface and would be best protected from injurious effects of too intense light if the rays are parallel to the leaf surface. This protection is attained in some sun plants by the erect or ascending position of the leaves or by their orientation in such a way that their surfaces face either east or west, as in so-called "compass plants." In other cases, as in many legumes, the leaflets, which in moderate light are spread out to receive the full-light income, fold either upward or downward in pairs when the light becomes intense (during midday in the summer) and thus place the leaf surfaces parallel to the incident rays of light.

In many cases in which protection of the chlorophyll apparatus cannot be obtained by the position of the leaves, the chlorophyll bodies change position within the leaf cells. During moderate illumination there is a tendency for them to be massed or grouped on the surface faces of the cells, while with too intense illumination they are shifted and arranged

along the walls vertical to the surface. This change of position may cause a deep-green color in the shade and a less intense or paler color in the intense light and will explain the so-called "shadow pictures" of shading organs upon brightly illuminated leaf surfaces.

With increase in the intensity of light, photosynthesis will increase up to a certain point, and then with further increase in intensity of light the photosynthetic activities remain about constant, but for a short time only. If the intensity becomes too great or the optimum is exceeded for too long a period, the construction of carbohydrate food becomes less and less active and may finally be checked entirely. During the exposure of green plants to ordinary illumination, the green pigment, chlorophyll, is being constantly oxidized, but it is being constantly reconstructed, hence the change is not evident. Under conditions of intense illumination, light-sensitive plants develop a pale or yellowish-green cast or even a bronzing of the leaves. Under such conditions, the chlorophyll is oxidized somewhat faster than it is reconstructed. This behavior is frequently noticed when plants that are normally shade-loving—*e.g.*, those that select the shaded forest as their habitat—are suddenly moved from glass houses with poor illumination to the bright sunlight of the open. The sensitiveness to intense light often varies with the age of the plant—*e.g.*, the seedlings of many trees are not able to withstand the direct sunlight, while older trees suffer no derangements from exposure to the same light intensity. In the more sensitive species, continued exposure to intense light may kill the protoplasm of cells in leaves, stems or fruits, and browning, burning or blighting of localized spots or more extended areas may be the final result. Specific sensitiveness to light and the conditions of moisture, temperature and light which have prevailed previous to the exposure to intense light have an important bearing on the type and degree of injury which results (see Unfavorable Water Relations, Chap. V; also High-temperature Injuries, Chap. VII).

In addition to suffering from too great intensity of light, it seems that some of our crop plants may suffer derangements because of too great a total light income—in other words, there may be a wrong balance between the total period of illumination or daylight and the total period of darkness. Apparently the normal life of the plant requires a certain balanced development of foods or certain nutritive or assimilative ratios, and a disturbance of these nutritive ratios throws the plant machinery out of gear. Too high a light income might cause an overproduction of carbohydrate food. Is it possible that in such a case the plant might suffer from "indigestion"?

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SUN SCALD OF BEANS

A spotting and streaking of beans in Colorado has been studied by Macmillan (1918) under the name of sun scald. This trouble appeared first when the pods were whitening.

Symptoms and Effects.—The first indications of the disease are very tiny brown or reddish spots upon the upper or outer valve away from the center of the plant. These spots gradually lengthen until they appear as short streaks running backward and downward from the ventral toward the dorsal suture. In 2 days, the spots have increased to areas of brown water-soaked tissue sometimes slightly sunken. If the spread has been rapid, the color is a good brown, sometimes tinged with red, extending over a majority of the exposed surface and sometimes over all of it. On some varieties, the entire exposed surface does not become covered, but spots 3 to 4 millimeters in diameter grow to be the largest, while new spots are constantly appearing. Often small spots coalesce into larger ones, giving them an irregular shape. Eventually this spotting may appear on the underside of the pod but always in lesser quantity (Macmillan, 1918).

Brown streaks on exposed stems and leaf petioles appear under the same conditions as the pod spotting or scalding, and the leaves also show a browning and death of the epidermis on both surfaces. The scalding

of the leaves may be mild and the result of light acting over a comparatively long period. It is stated that this leaf scalding results in indeterminate symptoms in some respects similar to mild mosaic.

The sun scald of beans causes little if any loss in a seed crop; but in severe cases, white-skinned varieties may have the seed coats slightly stained, thus affecting quality. No reduction in yield or lack of vigor has been noted. Undoubtedly, sun scald of beans is more common than reports would indicate, since it is very evident that it has been confused with bacterial blight. The disfiguring lesions on pods of string beans are of importance as affecting quality, and it seems probable that under certain conditions they may be the avenues of entrance of certain parasitic organisms. Observations indicate that while sun scald may be expected to be most severe in high altitudes, it is by no means confined to such localities. The sunburn of soy bean and cowpeas as it occurs in Arizona appears to be very similar to the sun scald of beans (Gibson, 1922).

Etiology.—While beans have been shown to be susceptible to heat injury (Macmillan and Byars, 1920), it has been proved that the type of injury described as sun scald is due to the action of light rather than to heat. In the first place, it has been shown that the tissue of the lesions is bacteriologically sterile; hence any causal relation of an organism is excluded. It has also been shown that pods subjected to the same temperature conditions were sun scalded when exposed to the full intensity of the sunlight, while shaded pods remained normal. By the use of artificial illumination from an arc light, streaking and browning of pods and bronzing and browning of leaves were produced similar in every respect to the symptoms and effects observed under natural field conditions. The artificial illumination did not cause sun scald if the light was obliged to pass through ordinary window glass, which is known to be absorptive of light of very short wave length. In the Colorado bean section, the altitude is 4700 feet, and during the heat of the summer days a relative humidity of 20 per cent or less is common. Under such climatic conditions, the light reaching growing plants will contain a much larger portion of the ultra-violet rays than the light of regions with higher humidity and lower altitude. In other cases, it has been shown that the ultra-violet rays have a killing effect upon active protoplasm and may apparently cause injury independent of the chemically active light rays. The conclusion has been reached that the sun scald of beans is caused by light of short wave length (ultra-violet). These conclusions are in keeping with the findings of other investigators who have recorded the injurious effects of light at high altitude upon the natural vegetation. If the conclusions are correct, sun scald of beans or of other tender species may be expected to be more frequent in the regions of high elevation and low humidity than in humid areas nearer to sea level.

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LODGING OF CEREALS AND OTHER CROPS

The lodging or falling down of cereals previous to harvest is a common phenomenon in many regions, while in others it is rather rare. No

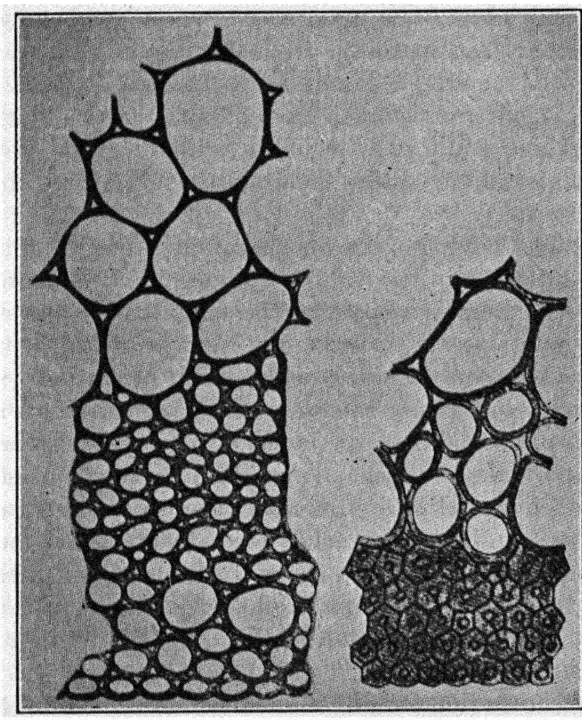

FIG. 62.—Cross-sections of normal and lodged rye culms. (*After Koch.*)

single cause of the lodging can be sighted, but the following may be noted as contributing factors: (1) excess of nitrogen, or at least a large amount of available nitrogenous food materials, which stimulate the plants to make a luxuriant growth with heavy foliage; (2) an abundance of moisture in soil and air, which promotes a succulent type of growth; (3) frost injury due to localized killing of the meristematic tissue at the base of certain internodes; (4) the attacks of insect pests or fungous invasions (straw breakers or foot rots), which are clearly parasitic in character or favored by frost injuries or traumatisms; (5) mechanical breaking or lodging due to the direct action of wind, hail or rainstorms.

While one to several of these factors may be operative in any single case, the most common cause of general lodging is a weak development of the bases of the culms or stalks as a result of the lack of light.

Symptoms and Effects.—The lodging due to partial etiolation of the basal portions of the culms involves a weakening and abnormal elongation of the second internode from the base, the lowest stem member being generally too short to bend. Under conditions which promote the weakened growth, the developing culms may fall, due to the bending at the second internode. Lodged grain may partially right itself, owing to renewed growth in other internodes as a result of the geotropic response induced by the prostrate position. The lodging of the grain interferes with the normal physiologic processes, especially photosynthesis and transport of crude food materials from the root system to aerial parts, and consequently the crop is lowered in both quantity and quality. In addition, the lodged grain is much more likely to suffer injury from semi-parasitic fungi and from such troubles as powdery mildew and rusts, while the difficulty of harvesting the grain that does develop is the cause of additional losses.

Etiology.—The lodging of cereals was at one time attributed to a lack of silicic acid (H_2SiO_3) in the soil, thus lessening the amount of silica in the cell walls and thereby weakening the supporting power of the culms. Such an explanation seemed possible until it was shown that there is but little difference between the silicic acid content of normal stiff straw and lodged straw and that a very small amount of silicic acid is sufficient for the production of normal plants. It has also been shown that the lowest internodes of normal plants are poorer in silicic acid than other parts of the plants, which would be opposed to the supporting function of the silicified cell walls. The silicic acid theory has received recent support (Phillips *et al.*, 1931) by studies showing that sodium nitrate fertilizer increased the lignin but gave a reduced silica content. This seems contradictory to the report of Welton (1928) that lodging is caused in part by the deficiency of lignin.

It has been shown that lodging is due to a modification of the structure of the lower internodes as a result of the interaction of many factors, with shading playing a prominent part. In thick stands of grain, the numerous culms with clustered and overlapping leaves prevent the penetration of light and also tend to prevent the circulation of the air and thus hold a surface stratum of more humid air. Both lack of light and increased humidity of the air promote the formation of delicate or thin-walled tissue of elongated cells, as opposed to the firm growth with well-developed mechanical tissue which is characteristic of well-illuminated structures surrounded by an air of more moderate humidity. Further support of the conclusion that shading is the most important factor in causing lodging has been obtained by the artificial production of lodging by

shading the basal parts of the plants, while the more distal parts were subject to normal light intensity.

The stems of normal cereals show a well-developed zone of thick-walled, prosenchymatous tissue below the epidermis, but in the partially etiolated basal internodes of shaded culms this cylinder of supporting tissue is poorly developed and other tissues do not have the normal supporting power. The greater length of these shaded internodes and the thinness of the cell walls make such stems poorly suited to withstand the strains they must bear, and they easily go down during wind- or rainstorms.

From extensive researches (Welton and Morris, 1931), the following conclusions may be presented:

1. Lodging results when there is a relatively low content of dry matter per unit length of culm.

2. Such culms are relatively small in diameter and are accompanied by a low carbohydrate-nitrogen ratio, resulting from hypernutrition, shading or high temperatures, the complex giving a relatively high proportion of vegetative growth.

Soils rich in available nitrogen and well supplied with moisture predispose to lodging, since tillering is increased and the heavy growth of foliage increases the shading. Thick seeding on moderately rich soil may sometimes lead to very general layering, because of the shading due to the very dense stand. With the modern practice of drill seeding in which the rate of seeding can be accurately controlled and adapted to the soil type, the danger of lodging is lessened.

Prevention.—The most important practices which may be followed for cutting lodging down to a minimum are as follows: (1) careful attention to the rate of seeding to suit the soil conditions so as to produce a stand which will allow penetration of light and a free circulation of the air; (2) attention to cultural practices, including rotations and the use of fertilizers so that excess of available nitrogen will not be offered; (3) the selection of varieties suited to the environment. Stiffness of straw and the ability to stand up are characters which vary with the variety, and improvement in this respect is one of the problems of the cereal breeder.

Other crops besides cereals frequently suffer from lodging—*e.g.*, field peas. Their stems are naturally rather weak; and when they do go down, rotting may cause additional damage if there is an abundance of moisture. In localities where the lodging tendency is very pronounced, a supporting crop is sometimes mixed with the peas. It is not uncommon to have too heavy stands in seed beds in the open or under glass. Under such conditions, the hypocotyl frequently elongates to several times normal length and the young seedlings fall over, and, as a result, irregular, twisted stems are formed. The partially etiolated stems are then more readily infected with damping-off fungi.

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PHOTOPERIODISM

In the general discussion of the effect of light upon the plant, it was pointed out that the response depends on three factors: (1) the intensity of the light; (2) the wave length or quality; and (3) the duration of the light action. Apparently the earlier work was devoted primarily to studying the effects of the first two of these factors, while it is only since the researches of Garner and Allard (1920) that the significance of the daily period of light in the growth and reproduction of plants has been appreciated. Their results were confirmed by Adams (1923), and, more recently, numerous other workers have contributed to our knowledge of the subject, with the result that there is a rapidly expanding literature with no end in sight until the photoperiodic response has been determined for the world's supply of plants!

Responses to Long or Short Periods of Illumination.—The length of daylight to which a plant is exposed is expressed in the type of growth which results. In nature, the establishment of a species in a given environment depends on its ability to flower and produce seeds, and this is possible only when the favorable length of day is presented. The favorable length of day for a plant is termed its *photoperiod*, and *photoperiodism* is the response of a plant to the relative length of day and night.

In the nature of their response to length of day, two extreme types of plants may be recognized: (1) *short-day plants*, or those which tend to a vegetative development with increase in stature when exposed to long daily periods of light and flower and fruit only when the light period or the length of day is suitably decreased; and (2) *long-day plants*, or those which show an altered development and fail to flower when subjected to short days but readily form flowers under the influence of suitably lengthened periods of daylight. Between these two extremes are plants less sensitive to the length of the daily period of light, and these are, therefore, able to make a growth in which flowering and reproduction are attained in the widest variations which occur in nature. The length of day is a factor of importance affecting the natural distribution of plants in different parts of the world. For example, if the daylight periods of an environment

during the season when moisture and temperature factors make growth possible are too short or too long to initiate flowering and carrying of the seed production to completion in a given species, the natural range of that species will be limited to those regions which offer the length of day which does make flowering and seed production possible.

The plasticity of plants under the influence of variation in the length of day and night to which they are exposed is very marked but finds its most striking expression in the effect upon flowering and fruiting, while various chemical and structural changes may be accompaniments. Flowering and fruiting may be retarded or accelerated, the type of vegetative growth modified to lead to gigantism or to dwarfing, while the laying down of reserve foods in bulbs, tubers or roots may be inhibited or seriously impaired. The anatomy of leaves or other plant organs may be changed, the production of fiber may be affected, while the ratio of flowers of different sex may be modified (corn) and in some cases even a reversal of sex may result (hemp).

In the cultivation of plants, the desired end may be production of a luxuriant vegetative growth, or *gigantism,* with an inhibition or delay of flowering or fruiting, while in a large percentage of our ornamental or crop plants the production of flowers or of fruit is the feature of commercial value. It is not possible within the space available to give any detailed discussion of the numerous illustrations of photoperiodism which have been studied, but a few cases may be cited which illustrate the practical application of the principle.

The growth of a plant like spinach may be noted. When planted in the early spring, a well-developed rosette is formed and flowering is delayed; but when planted later in the spring or in the summer, it makes a poorer vegetative growth and quickly sends up its inflorescence. While the temperature factor is also operative, the difference in the length of the day is, in the main, responsible for the changed behavior. It is not an uncommon experience for a farmer to make a mistake and seed a winter wheat in the spring, with the result that it makes a purely vegetative growth with the formation of rosettes that fail to produce culms and flowers. This has been explained by saying that it had the winter habit, but Wanser (1922) has recently called attention to the fact that the winter habit in wheat is but an expression of photoperiodism. He states that

> A proper adjustment of the daily exposure to light, independently of temperature, will control the type of growth in the winter-wheat plant, and by regulation of this factor it is possible to induce the jointing and the heading stages irrespective of season.

The poinsettia when exposed to the seasonal length of day will not flower until early winter; but by shortening the length of the day (10

hours), it may be made to form flowers and the brightly colored bract at any season of the year. The effect of length of day has been applied to a practical problem in tobacco culture. A variety of tobacco known as Maryland Mammoth is grown in southern Maryland. In that environs ment under suitable conditions, it makes a luxuriant vegetative growth-producing large plants with many leaves (sometimes more than 100) and, is, therefore, prized for its high yielding capacity. In Maryland, it does not flower or blossoms so late that no seed is matured—because the summer days are too long. It has been shown that it will flower and fruit in the greenhouse under the natural day length of winter, and seed is now obtained by growing the variety in southern Florida during the winter when the days are short. Onions are very sensitive to the light period (McClelland, 1928). Of four varieties tested—Prizetaker, Yellow Globe Danvers, Silver King and Bermuda White—only the last proved to be well adapted for growing in the tropics, where the maximum length of day is close to 13 hours. Under these conditions, the first three varieties either failed to produce bulbs, *i.e.*, remained in the spring-onion stage, or made only a partial or imperfect development.

Plants in their native haunts have become adjusted to the specific light periodicity of their environment, but in our agricultural and horticultural practice they may be subjected to light periods to which they are not suited: (1) by field planting in regions with too short or too long days; (2) by date of seeding in the field so that the optimum light periods are not experienced during the growing season; and (3) by cultures under glass which are independent of the season as far as temperature and moisture are concerned, but with seasonal variations in the length of day. Photoperiodism of plants has an important bearing upon the success of cultures under artificial illumination and emphasizes the fact that the desired results can be attained only with careful attention to the varying light requirements of different varieties. Failures to attain the desired ends in farm or horticultural practice may be due to a lack of acclimatization of our cultivated species with respect to the light relation or to our failure to understand the specific light requirement of the variety. Much of the recent work has brought out quite different responses of varieties, strains or "ecotypes" of the same species. This may be illustrated by winter, spring and summer varieties of lettuce or by the behavior of early, medium and late varieties of soy beans (Garner and Allard, 1930).

Etiological Relations.—Some considerations have been given to the way in which light duration induces the photoperiodic responses. It may be noted, first, that the length of day must affect the quantity of photosynthetic product formed, since photosynthesis is active and must continue during the period of illumination and cease during the periods of darkness. It seems also that the nature of these products and their utilization are modified by the length of day. In a recent paper (Garner,

Bacon and Allard, 1924), data were presented which "indicate that the light period in some way profoundly influences acidity relations, the form of the carbohydrate present in the plant and probably the water content of the tissues." Under certain conditions—*e.g.*, in gigantism of short-day plants—acidity of cell sap as measured by hydrogen-ion concentration is high, while in long-day plants exposed to relatively short days, the acidity remains at a relatively low level. More recently, Deats (1925) concludes that "the differences in the relative length of day and night influence the form of plant development by a change in the nitrogen-carbohydrate ratio." This view is also held by other workers; Tincker (1925, 1928) writes: "In general, there would appear to be a correlation between the C/N ratio and the behavior of the plant." The variation in the production and utilization of photosynthetic products is then the controlling factor. Photoperiodism is also held to be a response to the oxidation respiratory-synthesis ratio (Lubimenko, 1928). "In long-day plants, oxidation is proportionally greater than in short-day plants when compared with photosynthesis, so that these plants tend to use up carbohydrates rapidly in short days" (Tincker, 1929).

It has been shown that there is a definite localization of the effect of different periods of illumination; *i.e.*, when parts of plants are exposed to different periods of illumination, each part will show the characteristic response to its particular light period (Garner and Allard, 1925), but it has been demonstrated that the response of any particular part is still further localized in the growing points or apical buds (Knott, 1927).

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CHAPTER X

DISEASES DUE TO MANUFACTURING OR INDUSTRIAL PROCESSES

As a result of modern conditions in cities and the proximity of various types of industrial concerns, cultivated plants and native vegetation are frequently exposed to unfavorable factors which may operate either through the air or the soil environment. Material in dust form may fall on vegetation or on surrounding soil and cause injury by mechanical interference with life processes or by its ultimate toxic action. Various substances may be set free in manufacturing or industrial processes, and these may diffuse through the soil or the surrounding air and reach the root system or the aerial parts of plants either in gaseous form or dissolved in mist or raindrops. Mention may be made of sulphur dioxide, fluorine compounds, hydrochloric acid, chlorine, arsenic, sulphuric acid, nitric acid, and ammonia, the first being of outstanding importance. These may be by-products of industrial plants, as sulphur dioxide from smoke and smelters, or the main output of the plant, as illuminating gas. Very high toxicity to plant life, even when present in only minute quantities may be noted especially for sulphur dioxide and illuminating gas or its principal constituent, ethylene. Injuries may also result, especially to trees, from electric discharges from high-power transmission lines.

Cement-dust Injury.—Dust from cement mills has been shown to have an injurious effect upon the setting of fruit, according to the work of Anderson (1914). In the cases investigated, the dust came mainly from the stacks of the kilns and made an evident deposit on the vegetation within a radius of 2 miles. The reported reduction in the setting of fruit as a result of the "dust showers" was confirmed by experimental tests with cherries, pears and apples. Flowers artificially dusted with material produced from the cement plant showed very pronounced reduction in the percentage of fruit set, as may be illustrated by the reduction from 49.9 per cent in untreated apple blossoms to 5.95 per cent in the dusted blossoms. It was shown that the dust from the cement mills contained a large amount of alkaline, soluble calcium salts, and germination tests proved that pollen grains would not germinate in very weak solutions of these salts. The prevention of fruit setting by cement-mill dust may be explained as follows:

> When the dust falls on the fruit blossoms some of it goes into solution in the stigmatic secretions and pollen falling on the stigma will not germinate. Thus the flowers will not be fertilized (Anderson, 1914).

Magnesium Oxide Injury.—Crop injury has recently been reported (Sievers, 1924) as the result of deposits of magnesium oxide dust originating from roasters in which magnesite is calcined. The injury was confined largely to crops growing in an oval area 1 mile wide and 3 miles long, with the magnesite plant at its center. After the magnesium oxide dust is deposited on the soil, it is converted into basic magnesium carbonate by the action of carbon dioxide and water. Although not readily soluble in water, the magnesium carbonate in a soil solution containing carbon dioxide forms the more soluble bicarbonate, and thus the concentration of soluble salts is greatly increased.

The injury to wheat from the magnesium oxide deposits was of two types: (1) a yellowing and blighting of the leaves, beginning at the tips, with retarded growth and death of the plants in the more extreme cases; and (2) the mechanical interference with the emergence of seedlings, due to the mortar-like crust on the surface, this effect being very pronounced on land close to the calcining plant. The soil of the region beyond the influence of the roaster contained about 4000 pounds of magnesium, calculated as oxide, per acre-foot, while the amount increased gradually as the plant was approached, until at a distance of 100 feet it amounted to 56,000 pounds per acre-foot.

Injury from Tar Products.—The injurious effects of vapor or dust from tarred roads or of the fumes from melting tar compounds on vegetation have long been recognized. The observed effects have varied, depending on the character of the products and the amount of dust or vapor reaching the aerial parts of plants. The lesser effects noted are fading and spotting of leaves, while strong fumes cause the injured leaves to curl and shrivel, turn brown and fall. In the injured tissues, the cells are plasmolyzed, and the chlorophyll disappears.

A special study of the injurious effects of the fumes from tarvia, a tar compound used in building operations and in road construction, was made by Chivers (1917). The fumes from melting tarvia carried across a near-by garden covered leaves and stems with a greasy coating and killed various annuals and a number of perennials; peonies were killed to the ground; roses, brambles and currants were defoliated; while potatoes were dwarfed, and the yield reduced. All perennials within the affected zone showed the injurious effects in the growth of the following season. Experimental tests were made with a number of plants:

Leaves of begonias showed a characteristic sinking of the upper epidermis, at first in small isolated areas, which gave a peculiar pocked appearance to the leaves. The pock marks gradually became confluent and the entire area lost chlorophyll and turned brown. In the youngest leaves the first symptoms appeared as yellow spots, 3 to 6 millimeters in diameter, which when examined were found in each case to be an injured area immediately surrounding a multicellular gland.

Older leaves turned yellow over their entire surfaces and fell from the stem (Chivers, 1917).

The train of symptoms varied in different species subjected to the same concentration of fumes. Ferns "withered and died as if subjected to extreme heat." In geraniums, "the lower and older leaves turned yellow, those of medium age turned dark brown over the entire surface while the youngest and only partially unfolded ones showed dark-brown zones on their margins." It was further shown by tests with begonia leaves that the tarvia products were able to cause injury by penetrating the epidermis, the results being the same whether the fumes acted on the stomata-bearing undersurfaces or on the upper surfaces devoid of stomata. In a more recent study (Böning, 1926), tar-gas injuries to beets, cabbage and other truck crops were noted, but graminaceous species were said to be uninjured.

The principal volatile constituents of tar products are phenol, anilin, pyridin and pyrrol. Observations have indicated that the amount of injury from tarred roads depended on the amount of phenol in the compound used. Pyridin has been shown to cause severe injury to leaves subjected to its fumes, inducing plasmolysis in general, browning of tannin-containing cells but with no destruction of the chlorophyll. It seems probable that both phenol and pyridin are active in injuries produced by tar or tar products. The exact type of injury may be expected to vary with the type of tar products, the concentration of fumes or the amount of dust, the age of the plants or of the leaves and the species or varieties exposed.

Electrical Injuries.—The development of electric-light systems, trolley lines and high-power transmission lines has introduced a new element of danger to the trees of towns and cities. It is a matter of common observation in many sections that lightning causes much injury to trees, while numerous cases of lightning injury to field crops are on record. It is perhaps not so generally understood, however, that trees may suffer from electric discharges from transmission lines when these are too close. Either alternating or direct currents may cause injury, but the former is apparently less injurious. An electric current acting on a plant at certain strength—the *minimum*—may cause just perceptible stimulation, while the *optimum* causes the greatest stimulation. Beyond the optimum, plant activities are retarded, and at *maximum* strength death ensues. The maximum current necessary to cause death is exceedingly variable.

If it were not for the fact that trees are poor conductors of electricity, there would be much more injury when trunk or limbs make contact with live electric lines. The cambium, or the layer of cells containing active protoplasm, offers the least resistance, while the outer, dry, corky bark

offers the greatest resistance, with phloëm or inner bark, sapwood and heartwood occupying an intermediate position in the order recorded. The high resistances shown by the tissues of a tree are capable of cutting down rather high currents to an insignificant amount. Injury results, however, in wet weather

> . . . when the tree is covered with a film of water, which provides favorable conditions for leakage, the current traversing the film of water on the tree to the ground. The result of contact of a wire with a limb under these conditions is a grounding of the current and a burning of the limb due to "arcing." The vital layer and the wood become injured at the point of contact, resulting in an ugly scar and sometimes the destruction of the limb or leader (Stone, 1914).
>
> The alternating-current systems employed for lighting purposes vary greatly in their potential. Cases of burning from alternating currents are more numerous than those from direct currents because trees are brought into more frequent contact with the wires, and, owing to the higher potential, more leakage is likely to occur. The high- and low-voltage lines may vary from 100 to 100,000 volts. The high-tension systems are invariably constructed across country, and are naturally not brought into very close proximity to shade trees. No injury whatever occurs from the low-voltage (110-volt) lines, but the lines of higher potential found on streets constitute a source of danger to trees. The higher the electrical potential the more dangerous the wires become to trees, for, owing to the lessened effectiveness of the ordinary insulation, more leakage occurs, and consequently greater opportunity for burning (Stone, 1914).

Alternating currents cause only local injury to trees, the lesions appearing only near the point of contact with the wire. The tree is not killed, but limbs may be so badly burned as to cause serious disfigurement.

"Most of the *direct currents* affecting trees are those used for operating electric railroads. Trolley feeders may be at 500 to 550 volts" (Stone, 1914). Localized burning, similar to that resulting from alternating currents, may take place, but under certain conditions large trees may be killed by direct currents used in operating electric railroads. In case of death of trees from direct-current electricity, the rail is positive and the overhead feed wire negative, constituting what is called a "reversed polarity." It is the more common practice, however, to have the so-called positive current traverse the overhead wire, but "reversed polarity" is used at times. In a typical case, the escaping current had

> . . . burned and girdled the trunks for a distance of 5 to 10 feet from the base, the point of contact of the feed wire with the limb 18 to 20 feet above showing little or none of the characteristic local burning effects usually observed in ordinary cases of grounding.

The more extensive burning in cases of reversed polarity is favored by the moisture conditions of soil and bark at the base of the tree offering a reduced resistance. The general result will be slight burning at the point

of contact of the overhead wire, with extensive burning at the base, although the injured zone may be variable in height. The cambium is killed, and the bark may become loose and fall off. Injured trees generally stand fairly close to the rails, although those more distant may be killed if there is a ground connection.

As a general principle, trolley or electric-light wires should not be allowed to come in direct contact with the trunk or limbs of trees. If, however, contacts cannot be avoided, proper insulation of the wires should be provided.

Injury from Illuminating Gas in the Soil.—Herbaceous or woody plants growing in streets, yards or greenhouses may be injured by the leakage of illuminating gas into the soil. The greatest amount of damage

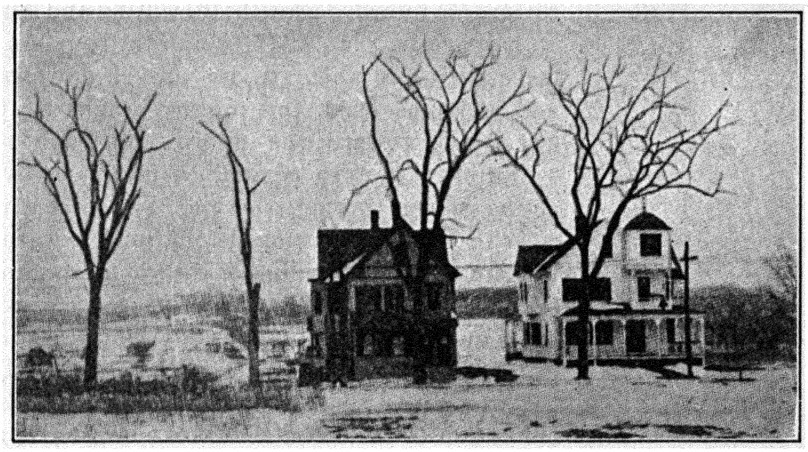

FIG. 63.—**Large elms killed by escaping illuminating gas, 1½ years after leakage occurred.** (*After Stone, Mass. Bul.* **170.**)

occurs in shade trees of street or lawn. Injury to trees from leakage of gas into the soil from defective joints or broken pipes is a problem with which every large gas-producing company must contend. Much of the gas that is manufactured is unaccounted for, part of the discrepancies being due to defective meters or incorrect readings, but more or less actual leakage does occur. Slight leakage of gas into the soil through a long period may cause a slow poisoning of the roots of near-by plants, while with more pronounced leakage acute injury may follow very rapidly.

Severe injury to trees in several German cities was reported by Girardin as early as 1864, and his conclusions have been confirmed by numerous investigators since that time. Special studies of gas injury due to leakage into soil have been made in this country by Stone (1913, 1916), Harvey and Rose (1915) and Doubt (1917), while more recently Wehmer (1917, 1918) in Germany has made a general study of injury due to illuminating gas.

The effects of illuminating gas are variable, depending on the age and stage of development of the plant, the variety or species concerned and the duration of action and concentration of the gas. Some of the effects of illuminating gas are as follows: (1) the inhibition of germination of seeds; (2) abnormal curvatures and swelling of young rootlets, due in some cases to hypertrophy of cortical cells and in others partly to hypertrophy of cells and partly to increased cell division; (3) the formation of proliferation tissue in the cortex of woody stems below the surface of the ground; (4) the disappearance of starch from the cells of the root cortex; (5) retarded growth; (6) wilting and death of herbaceous plants; (7) epinastic response of leaves, causing them to be turned downward and in some cases the upward rolling of the leaf blade toward the midrib, although this symptom is not so general as in the cases of action of gas through the air; (8) the death of trees or shrubs following defoliation or death of the leaves. The swelling of the young roots and the disappearance of starch from the cortex of roots are responses to rather low concentrations of illuminating gas. The roots of trees killed by gas frequently show a characteristic bluish discoloration of the wood, but this symptom is not diagnostic, since it is known to follow death by other agencies. The production of proliferation tissue by roots is a feature which may be used in diagnosing gas injury. Root hypertrophies occur when the foliage shows no effects; hence it is suggested that an examination of the roots of adjacent plants be made when a tree has died from suspected gas leakage. The cortex of young gassed roots is sometimes three to four times the thickness of that of normal roots. The characteristic hypertrophies might not show on the root system of a dead tree, if it had been subjected at once to a high concentration of gas, while they might appear on roots of near-by uninjured plants.

FIG. 64.—Effects of illuminating gas on elm tree 1½ years after the leakage occurred. (*After Stone, Mass. Bul.* **170.**)

It should be noted that most of the symptoms of gas injury are of a general character, such as would result from the failure of the root system to function from any cause. Asphyxiation, freezing injury to roots,

collar rot, etc., might produce very similar results in trees. Trees killed by gas show a browning and disintegration of cambium, phloëm and cortex, especially at the base of the trunk, the dead area advancing upward until the whole tree is involved. The wood also darkens, and there is an increased brittleness with rapid deterioration. Gas-killed trees are quickly invaded by various saprophytic fungi, which complete the work of destruction.

The injury to roots from the leakage of illuminating gas into the soil may be due to (1) true asphyxiation, as a result of the displacement of the soil air by the gas; and (2) the toxic action of the gas constituents. It seems probable that both are factors of importance. Illuminating gases are of variable composition, but they always contain substances which are toxic to roots. Ethylene gas is always present, and tests with this alone have shown that it will produce practically the same effects as the composite illuminating gas (Harvey and Rose, 1915). More recently, Wehmer (1918) has advanced some evidence that hydrocyanic acid is the most toxic constituent of illuminating gas.

Plants show considerable variation in resistance to gas poisoning. Most deciduous trees are sensitive to gas in the soil, as has been shown by tests with Carolina poplar, elm, ash, maple, catalpa, apple, pear and others. Conifers are much more resistant than deciduous trees and may recover after injury has become apparent if the leakage of gas is stopped. On the contrary, deciduous trees which show clear-cut symptoms of gas poisoning rarely if ever recover.

The certain diagnosis of gas injury is rather difficult and requires the services of an expert. It may be pointed out, first, that young trees, at least, may be killed by the leakage of illuminating gas in an amount too slight to be detected by the odor. This possibility should be kept in mind when diagnosing cases of suspected gas poisoning. Stone (1916), who has made rather extensive studies of gas injury through a period of years, has placed great emphasis on the odor of the tissue of gas-killed trees for the recognition of the cause. According to his experience:

> There will always be found in trees killed by gas peculiar characteristic odors difficult to describe and more easily recognized, at least above the ground, after a tree has been dead for a few weeks or a month.

The etiolated-sweet-pea-seedling test can be used effectively in demonstrating the presence of illuminating gas in the soil when the odor of gas is not distinguishable (Knight and Crocker, 1913; Harvey and Rose, 1915). Seedlings may be grown in Petri dishes until several centimeters high and then placed under inverted cans on the soil supposed to contain illuminating gas. Strong concentrations of gas will cause a cessation of growth, while minute traces of it will induce diageotropic growth of the epicotyls; *i.e.*, they will grow in a prostrate or horizontal position. It is

claimed that this test exceeds many fold the delicacy of any chemical test.

The prevention of leakage of gas into the soil is given special attention by gas manufacturers, since corporations are frequently called upon to pay damages for killed trees. Stone (1916) has originated a special device to lay over gas mains to convey the leaking gas to certain points aboveground. When gas leakage is discovered, it should be stopped at once. Trees, except conifers, already visibly affected are not likely to recover, but any possible recovery will be supported by digging up the soil so as to allow the escape of the gas. New trees should not be set in the place of gas-injured ones until the soil has been thoroughly aerated.

Injury from Illuminating Gas in the Air.—For plants grown in the open, illuminating gas is not likely to accumulate in the air in sufficient amount to cause injury by direct action on the aerial parts, but severe injury has been noted in house and greenhouse culture. Crocker and Knight (1908) showed that ethylene gas was the probable poisonous constituent, causing injury when present in very minute quantities. Carnations proved especially sensitive, buds being prevented from opening by 1 part of ethylene to 1,000,000 parts of air, while 1 part of ethylene to 2,000,000 of air caused open buds to close. Under house or greenhouse conditions, only small quantities of gas are likely to be present in the air (White, 1926), but many glass-house plants may be injured by these small quantities of gas.

The types of responses induced by illuminating gas or by ethylene alone are as follows: (1) *Yellowing or falling of leaves:* with high concentrations, abscission may take place without any antecedent yellowing. Old plants are more sensitive than young ones, and old leaves drop more quickly than young ones. The falling of the leaves is probably due to the formation of an abscission layer. (2) *Rigor and loss of irritability.* (3) *Bud and flower injury:* buds may fail to open or open and drop their petals (roses), or open flowers may close and blight. (4) *Epinasty of petioles.* The drooping and twisting of leaf petioles are especially pronounced in certain species under suitable concentrations of gas. In Lycopersicum and Salvia, complete spiral coils may be formed. (5) *The formation of proliferation tissue.* Soft spongy tissue may be formed at the lenticels in certain species, or at the leaf scars, as in Lycopersicum, or at more extended regions along stems. (6) *The forcing of latent buds:* this response has been noted especially for roses (Zimmerman *et al.*, 1931). According to Doubt (1917), tomato, scarlet sage, sensitive plant, castor bean and Jimson weed are admirably adapted for use as test plants for illuminating gas in greenhouses, as "the response in each is definite, striking and not easily mistaken." Fifty parts of illuminating gas per million of air caused epinastic growth of the petioles of all of these plants. Healthy specimens of any of these test plants grown in pots and bearing

6 to 12 or more leaves may be placed at various locations throughout the greenhouse and left 24 to 48 hours with poor ventilation.

With only a trace of gas present in the air, the epinastic response of the leaves will be very noticeable if the plants are compared with normal plants without gas. This bending down of the leaves will increase with the concentration of the gas present in the air. All of these plants will drop their leaves with a concentration below the limit of the odor of gas. The older leaves fall first, the younger leaves being retained until there is 1 part of illuminating gas to 1000 of air.

The etiolated-sweet-pea-seedling test previously mentioned may also be used for detecting the presence of traces of gas.

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SMOKE INJURY

The atmosphere of industrial centers in the large cities and surrounding smelters in outlying districts is frequently polluted with various gases or dust materials which have injurious effects upon neighboring vegetation. The most important sources of injury are the products of the incomplete combustion of coal and the gaseous or solid wastes resulting from the smelting of ores. Investigations have shown that dust and metallic fume are elements of minor importance as far as damage to vegetation is concerned, in comparison with the gaseous constituents of smoke. Under open or field conditions, the most injurious gas is sulphur dioxide (SO_2), which is formed in the burning of coal and in the smelting of sulphide-containing ores. Smoke injury as here considered will then be confined very largely to the effects of SO_2.

Historical Statement.—Smoke injury to vegetation has been recognized for many years, but it was not until 1866 that SO_2 was shown to be the important toxic agent. Very positive proof as to the extreme toxicity of sulphurous acid (H_2SO_3) was presented by Schroeder in 1872. Since that time, numerous investigations and reports have been issued covering various phases of the smoke-injury problem. The importance of smoke injury in our modern life may be judged in part by the appearance of three outstanding volumes: (1) "Die Beschädigung der Vegetation durch Rauch und die Oberharzer Huttenrauchschäden" (Schroeder and Reuss, 1883); (2) "Die Beschädigung der Vegetation durch Rauch" (Haselhoff and Lindau, 1903), a handbook of over 400 pages with a bibliography of 126 titles; and (3) "Die Beschädigungen der Vegetation durch Rauchgase und Fabriksexhalationen" (Stoklasa, 1923), a very detailed treatise with over 400 literature citations.

In this country, the smoke nuisance in a number of the larger cities has prompted special investigations, those of Clevenger (1913) and McClelland (1913) of the Mellon Institute in Pittsburgh being noteworthy. The smoke problems of English industrial centers like Leeds and Sheffield have been given careful study (Crowther and Ruston, 1911; Ruston 1921). The injury from smelter fumes has been a problem in various parts of the United States and has attracted the attention of both chemists and plant pathologists. Special attention may be made of the work of Heywood (1905, 1908) in the vicinity of the famous smelter at Anaconda, Mont., and the report of the Selby Smelter Commission (Holmes *et al.*, 1915) which investigated the conditions surrounding the plants of the Selby Smelting and Lead Company, in Solano County, California. The department of smoke investigations for the American Smelting and Refining Company in Utah has continued the work started by the Selby Smelter Commission but on a much more elaborate scale. Other companies have followed their example and have given scientific study to their local problems but on a more modest scale.

Symptoms and Effects.—Three different types of injury from sulphurous acid in the air are recognized: (1) *acute*, when the amount of gas is abnormally high, being characterized by the rapid bleaching or disappearance of chlorophyll and in most severe form by the death of the entire plant; (2) *chronic*, when small quantities of SO_2 are generally present, leading to a general depression of physiological processes, including photosynthesis, metabolism, cell division, etc., with retarded growth,

exhausted food reserves, failure to blossom and set fruit, early leaf fall in deciduous forms or fall shedding of leaves by evergreens, ending ultimately in death; and (3) *invisible*, or the reduction of growth increments not visible to the naked eye but expressed by yields or by modified composition shown by careful measurements or by chemical analyses.

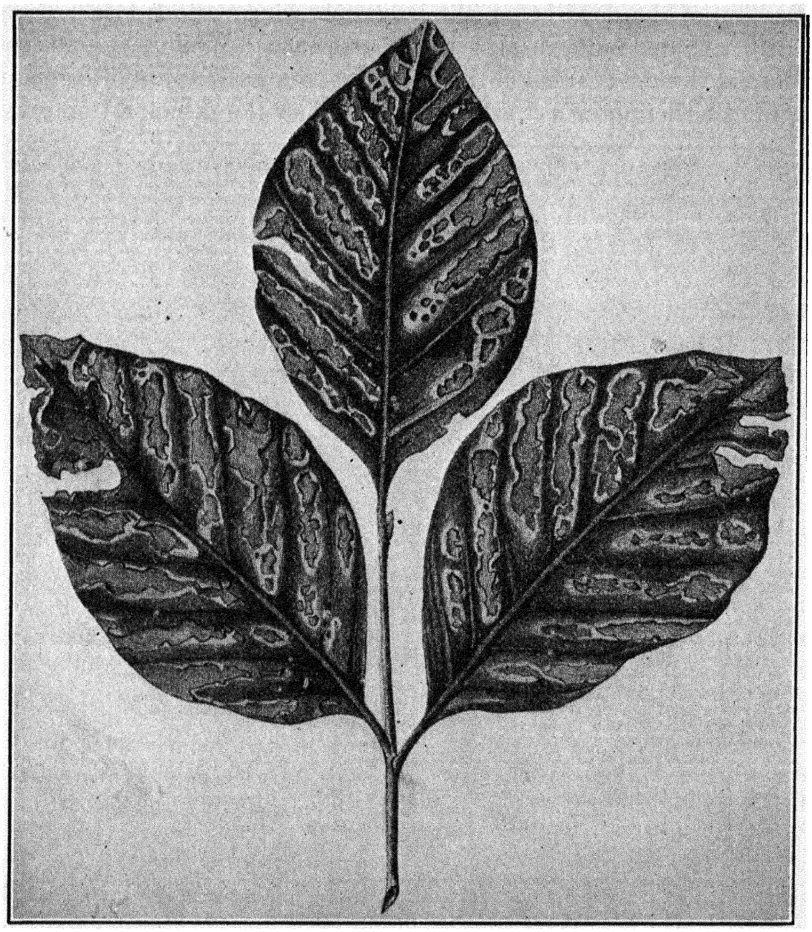

FIG. 65.—Beech leaves showing SO_2 injury. (*After Schrœder and Reuss.*)

There is no hard and fast line between acute and chronic injuries, but acute injuries are first indicated by characteristic changes of the chlorophyll-bearing structures. The response is somewhat different for the conifers, deciduous trees and shrubs and herbaceous forms. Acute injury in many *conifers* is marked by a wine-red coloration of the needles, sometimes for their entire length or sometimes first at either base, tip or middle. The needles then turn brown, shrivel and fall if the action of the gas continues or is sufficiently severe. The amount of discoloration and death of leaves is variable, but in general their length of life is

shortened, and trees close to sources of smoke frequently retain only the needles of a single year. In *deciduous trees and shrubs,* the most common result is the appearance of yellowish-brown to dark-brown dead areas in the intercostal areas of the leaves, while the mesophyll adjacent to the prominent nerves remains green for the longest time. Because of this localization of the dead areas, the leaves showing various types of venation will exhibit quite bizarre color patterns. While the intercostal location of the dead areas is the most frequent, the injured leaves may sometimes either show a dead brown border or the discoloration may be

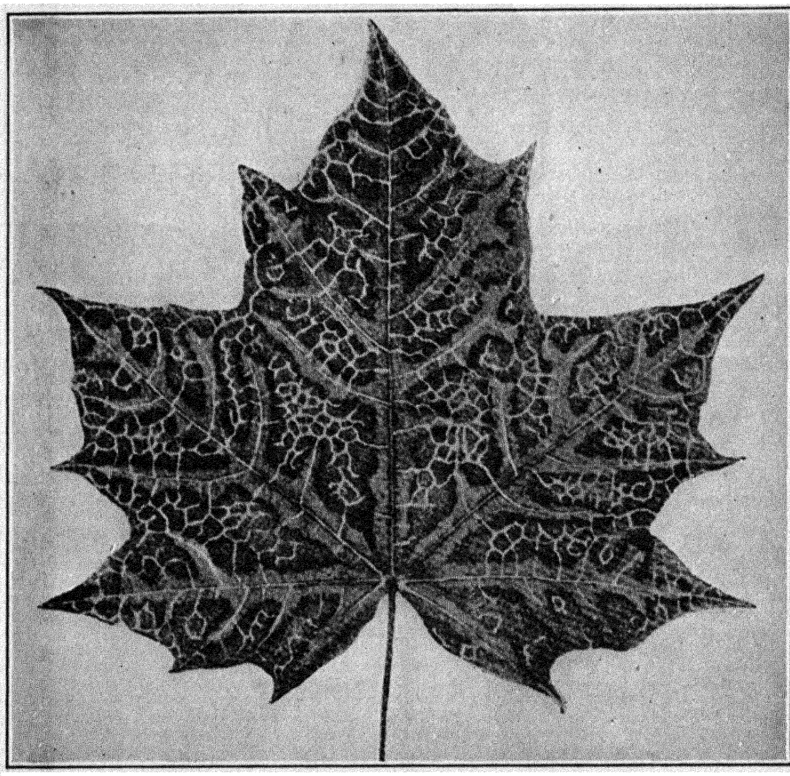

FIG. 66.—Maple leaf showing SO_2 injury. (*After Reuss.*)

confined first to either tip or base of the leaf blade. In species of Prunus or other forms showing a natural tendency to shot hole, the dead areas may fall away, leaving perforations. In *herbaceous plants,* the injured tissues may show all gradations of color from reddish brown or almost black to light yellow or straw color, with the lighter colors predominating. Injury to wheat before blossoming time is marked by the reddening of the leaf tips, which later turn yellow and finally become almost white. Other cereals and many grasses may show only a yellowing and bleaching of the leaves, beginning at the tips. Alfalfa and related legumes show extremes of bleaching or a clean, white appearance of the affected parts, the dis-

coloration advancing from the tips or margins of the leaflets so that an irregular green area may be left along the midrib. Lupines may show a dark-brown or almost black discoloration beginning at the tips of the leaflets, the sugar beet reddish-brown discoloration with a tendency to appear in the intercostal areas, while the potato may show reddish-purple tints similar to those characteristic of leaf roll. It should be pointed out in this connection that the various leaf discolorations which accompany SO_2 injury are in no way diagnostic characters, for very similar effects may follow injury from other agencies, such as drought, frost, sun scald, etc.

Chronic injury was first recognized for conifers and, according to some investigators (Wislicenus, 1914), does not occur in broad-leaved trees and general crop plants. This view is refuted by Stoklasa (1923) and other recent workers. The symptoms of chronic injury in conifers are not clearly defined, discoloration of the leaves being a minor symptom, while the injury is indicated mainly by the three following disturbances: (1) a shortening of the life of the needle leaves; (2) low increments of growth as marked by narrow annual rings; and (3) stag head, or bare terminal branches. In smoke zones, the life of spruce needles may be 2 to 3 instead of 4 or 5 years, and the life of fir needles may be reduced to 4 to 5 years in chronic smoke injury as compared with 10 to 12 years in normal trees. The exact effects will, of course, vary with the concentration of SO_2 to which the trees are exposed and with other modifying factors.

Chronic injury to field crops may be illustrated by the results obtained by Stoklasa (1923) with barley, wheat and sugar beets. All crops grown in the smoke zone showed some foliage injury, reduction in size and vigor of plants, premature ripening and reduced yields as compared with the controls. The barley that was harvested showed a lowered starch content, and the sugar beets a reduced storage of sugar. Chronic injury is well illustrated by some cases cited by Ruston (1921) from observations in Leeds, England. In portions of the smoke zone, bulbs flowered the first year but would not bloom the second season or thereafter, while lettuce and cabbage would grow but would not head. The behavior of the common privet under these conditions is also of interest: 3 miles north of Leeds it is evergreen and flowers, 2 miles north it is still evergreen but does not flower, 1 mile north but a few of the leaves are retained during the winter and in the center of the city the leaves fall in January, while in the heart of the industrial center they fall in November. Dwarfing and early leaf fall of other broad-leaved species were also noted, as may be illustrated by the fall of ash leaves on Sept. 18 in the industrial district and their retention until Nov. 1 in the district 3 miles north. Change of color of flowers was also noticeable, with paler tints as the industrial district was approached, with blues and reds tending to white and the bronzes to yellow. The scarlet of geraniums became streaked with

purple, and the blood-red wallflower streaked with yellow. Chronic injury has also been noted for tree fruits and grapes (Stoklasa, 1923). The former set little or no fruit, and in the latter the clusters were reduced in size, and by the month of August visible reddish-brown flecks appeared in the leaves.

The existence of *invisible injury* has been disputed by some workers, but Stoklasa and other German investigators have pointed out the serious injuries which may occur in the absence of either acute or chronic symptoms. The action of the SO_2 has a depressing effect on photosynthesis and other physiological activities, resulting in a general slowing down of constructive metabolism. Some of these depressions are reflected in the lowered starch content and reduced weight of cereals, reduced sugar content of organs in which this carbohydrate is normally stored, a low rate of protein to non-protein sulphur, reduced or lowered viability of seed and decreased hardiness or increased susceptibility to the inroads of some parasites. Ruston (1921) cites the case of oats grown in the outlying districts of Leeds, which had a germination of 98 per cent, while seed from the industrial center showed only 17 per cent viable. The effect on hardiness of winter annuals is shown by the winter killing of spinach, cabbage and wallflower in the regions around Leeds in which the annual deposit in soot per square mile was 200 tons or more, whereas beyond this area in regions with an annual deposit of 100 tons per square mile fall planting was uncertain, while the same plants were winter hardy in the more outlying districts.

Emphasis has been given to the effect of SO_2 on flower structures and fruiting by the recent work of Döpp (1931). This may be *indirect*, by injuries to the foliage, or *direct*, involving effects on the formation and maturing of anthers and pollen or pathological changes in stigmas, styles, egg apparatus and even in young embryos following fertilization. The effect on the germination of pollen and the growth of pollen tubes was studied in some detail. Under conditions of moisture favorable for germination, the pollen tubes were retarded in growth and frequently ruptured at the tip, with dilutions even of 1–1,000,000 causing injury. These effects will explain some of the previously reported cases of sterility or the production of seed with low viability.

The indirect effect of smoke pollution as a result of soil changes has been emphasized by some investigators (Ruston, 1921; Ewert, 1924):

The acidity of the smoke will deplete the soil of its calcium carbonate and in so doing will modify to a large extent the number and activity of the soil flora. The greater the acidity of the soil the smaller the number of bacteria present in the soil and the less their activity, the nitrifying organisms being found to be most susceptible. Bacteriological analyses were presented to show that the detrimental effect of the smoky atmosphere upon plant growth is partly due to unfavorable changes in the soil, such as the steady depletion of the stock of

calcium carbonate and the inhibition of the activities of the nitrogen-adapting soil flora.

Smoke pollution of soils affects the root system, "plants grown in soil that has been exposed for long to such pollution being marked by an almost entire absence of root hairs and fibrous roots" (Ruston, 1921).

Etiology.—It has been repeatedly demonstrated that SO_2 is the most important polluting agent in the smoke of industrial centers or from smelters, and experiments have shown the extreme toxicity of this compound for growing plants. SO_2 is a colorless gas, with a characteristic suffocating odor, and is 2.21 times heavier than air. It has a bleaching action upon many organic coloring matters, as may be illustrated by its effect upon chlorophyll and the pigments of flowers. In the presence of water, it behaves as though *sulphurous acid* (H_2SO_3) were formed, but this substance has never been isolated. It may be still further oxidized to form sulphuric acid (H_2SO_4), and some of the spotting of leaves and flowers in smoke zones is due to the action of this acid.

The average person cannot detect SO_2 in the atmosphere by the odor when the amount is below 3 parts per million of air. Injury to plants may result, however, when the amount is much less than can be detected by odor. The concentrations required to do injury to plants, according to published reports based on field analyses and experimental tests, vary from 1 to 40 parts SO_2 per million parts of air. The toxicity danger point will vary for different plants and will be affected by their stage of development but will also be modified by environmental factors. The toxic limits for some trees have been given as follows: oak, 1–720,000; pine, 1–500,000; and beech, 1–314,000. Roses show visible injury with concentrations of 1–250,000 to 500,000. According to Holmes *et al.* (1915), "a concentration of 10 parts SO_2 per million parts of air is necessary to produce injury to growing grain." The figures are sufficient to emphasize the extreme toxicity of SO_2.

There has been some difference of opinion as to the avenue of entrance of the SO_2, some of the earlier workers contending that it penetrated the epidermal walls as readily as through the stomatal openings, but Weiler (1905) working with deciduous trees and Neger (1914) with conifers have shown that the penetration is almost exclusively through the stomata. This will hold true for fully formed leaves with well-developed cuticle, but in immature tissues an appreciable absorption will take place through the epidermal wall. The conclusion is based on the observations that any treatments which cause a closing of the stomata during the period of exposure to SO_2 will either prevent the injury entirely or very greatly retard it.

The sensitiveness of conifers to smoke injury is a matter of common observation, and this has been attributed in large part to the clogging of the stomata by soot and tars, the deeply sunken stomatal pits being

especially favorable to the collection of these materials (Bakke, 1914). Numerous microscopic examinations of conifers from smoke zones have shown heavy black deposits overlying the guard cells, and these deposits were believed to be tar or soot originating from the smoke. This subject has recently been investigated by Rine (1924), who has found the same deposits in the stomata of leaves far away from any smoke zones. He concludes that the black stomatal deposit is a natural product of the leaf in xerophytic conifers in the nature of a wax which is finely granular and permeable to gases and therefore that no relation exists "between wax in the stomata and the high sensitiveness of certain conifers to smoke, except that as a factor inducing xerophytism the wax may lower the resistance of the tree."

The injurious effect of SO_2 is due to its diffusion through the stomatal openings into the interior intercellular spaces and its penetration into the living cells, where it interferes with the physiological functions of cytoplasm, kinoplasm and chlorophyll or in acute injury completely inhibits the life processes. This injury depends, in part, on the affinity of the SO_2 for oxygen and its tendency to combine with aldehydes, substances formed especially in chlorophyll-bearing cells. SO_2 will, therefore, be expected to have a profound influence upon the photosynthetic processes, or the construction of carbohydrate food. Microscopic tests show that leaves injured by SO_2 will contain either no starch grains or very few, and this is in accord with the influence of SO_2 in reducing the carbohydrates in seeds or vegetative storage organs. The reduced supply of available carbohydrates will, therefore, materially reduce the constructive processes or the growth of the injured plants.

The presence of small quantities of SO_2 has a marked effect on the process of transpiration, or water loss. First, there is an increase in the rate of transpiration, to be followed soon by a lower water loss than takes place in normal plants. Since transpiration is something of a measure of growth, retarded growth should be expected in SO_2 poisoning. This effect on transpiration may be noted in the different response of healthy shoots and those injured by SO_2. In healthy shoots, the leaves draw moisture from the young succulent stem, which will wilt and droop, while under exactly the same conditions the smoke-injured shoot will remain turgid and erect.

SO_2 injury is increased by light, moist air and high temperature. When plants are exposed to a toxic concentration of the gas, specimens placed in the light are injured, while others treated in every way the same, except for being kept in darkness, will show no injury. The amount of injury for a given concentration of gas is proportional to the intensity of the illumination, or it might be stated perhaps with more exactness that the greater the photosynthetic activity the greater the injury. This being true, the SO_2 injury is negligible during the night periods and sinks

to a minimum during the winter when plants are dormant. It has frequently been demonstrated that injury occurs sooner in warm, moist weather than under cool, dry conditions. Injury to young sugar beets has been noted in moist, warm weather in contrast to slight or no injury when dry weather prevailed.

The Diagnosis of SO_2 Injury.—The determination of the presence or absence of SO_2 injury in a suspected case is not a simple matter, since symptoms alone are not conclusive proof of the type of injury. The absence of known parasitic troubles should be given consideration, but their presence should not be taken too seriously as indicating the absence of SO_2 injury. In connection with the symptomology, certain tests may

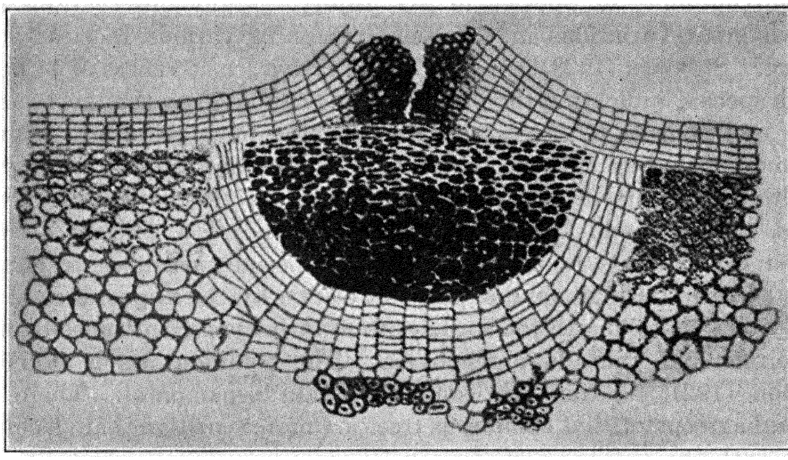

FIG. 67.—Section showing effect of SO_2 on sublenticular tissue. (*After Dr. Kupka.*)

be employed which will aid in arriving at a correct diagnosis. Some of these are: (1) an analysis of the air at representative stations for its SO_2 content for comparison with known toxic concentrations; (2) analyses of the leaves of plants from smoke zones for their SO_2 content, for comparison with similar leaves from normal habitats; (3) the behavior of special plant indicators, or the use of catch plants; and (4) the study of lenticel sections from sensitive species for the presence of a sublenticular area of brown dead tissue walled off from the sound tissue by a cork layer (Neger, 1919).

Numerous analyses of smoke-injured foliage have shown that the SO_2 content is frequently two to four times greater in the injured leaves than in normal leaves of the same species. Since, however, the SO_2 content of plant tissues is influenced by the soil composition, conclusions based on leaf analysis must be made with care. Lichens are especially valuable as indicators of SO_2 injury, since they are especially sensitive. They are rare or absent from the trees of cities, due largely to the SO_2 content of the air, but it may be noted that their prevalence is affected by other factors

also. The epiphytic alga Pleurococcus has also been cited as an indicator, but it is less sensitive than lichens. Several different cultivated plants have been recommended as catch plants for cultivation in areas in which smoke injury is suspected: (1) beans (*Phaseolus vulgaris*) by Sorauer; (2) species of Polygonum or Rheum by Haselhoff and Lindau; (3) a variety of grapes the leaves of which turn red when injured by SO_2 (Weiler); and (4) *Lupinus angustifolius* by Stoklasa. The chlorophyll of lupine, which is especially sensitive, is decomposed by 0.0004 to 0.0008 volume per cent of SO_2. The essential in the catch-plant method is the use of a sensitive species in which injury may be determined with certainty by botanical or chemical means.

Susceptibility of Species to SO_2 Injury.—Many observations have shown great variations in the sensitiveness of various species to SO_2 injury. Stoklasa (1923) gives the comparative sensitiveness of 44 herbaceous species, indicating the lupine (*L. angustifolius*) as the most susceptible and chicory (*Cichorium intybus*) as the most resistant. Various legumes, including clovers, beans, peas, lentils and alfalfa, occupy the sensitive end of the list, grasses and cereals an intermediate position while beets, potatoes and Brassica species stand near to chicory. Among garden ornamentals, roses are reported as especially sensitive, responding to 0.00026 to 0.00058 volume per cent of SO_2 by the appearance of reddish-violet spots due to the formation of anthocyanin in the cells of the epidermis and palisade parenchyma. The common iris is noted by Ruston (1921) as the most smoke-resistant ornamental. There is a general agreement that coniferous trees are more sensitive than deciduous ones, with the exception of the ash (*Fraxinus excelsior*), which Stoklasa places at the sensitive end of a list of 30 tree species. The spruce (*Picea excelsa*) is noted as the most sensitive of the evergreens, with the common yew (*Taxus baccata*) the most resistant. The maple (*Acer campestre*) is the most resistant of the deciduous trees, while the fruit trees—cherries, apples, peaches and apricots—occupy an intermediate position between the conifers and most other deciduous trees, although the birch (*Betula alba*) is more sensitive than some of the fruit trees.

Control or Prevention.—The prevention of injury to the natural vegetation or to cultivated crops is largely in the hands of the agencies which are responsible for the smoke production. The methods for the mitigation of the smoke nuisance are based on either the retention of the injurious substances or their dissipation in such dilute form that the concentration will never reach the danger point. Some of the suggested methods of retaining the noxious gas are condensation methods, decomposition with hydrogen sulphide with the deposition of sulphur, absorption of the acid gases by basic materials or washing the gaseous output with water. If the commercial demand justifies, the SO_2 output may be utilized as a source of sulphuric acid. The devices for diluting the SO_2

before it reaches vegetation are high smokestacks, numerous small stacks or some modification of this principle or special devices for diluting the SO_2 with air or combination of diluting and deacidifying. When it can be proved that industrial plants are responsible for crop damage as a result of SO_2 or other exhalations, they are liable for damage.

When the elimination of the smoke injury is not or cannot be accomplished, a certain measure of relief can be obtained by the selection of more resistant species for cultivation.

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CHAPTER XI

DISEASES DUE TO CONTROL PRACTICES

The treatment to save crops from losses due to diseases or to insect pests is sometimes as productive of injury as the disease or pest. In treating seeds or plants with either fungicides or insecticides, by spraying, dusting, steeping or fumigating, chemical elements or compounds are employed that are poisonous or toxic to fungi, bacteria or insects, and these same preparations may be toxic or have injurious effects upon our crop plants or upon the commercial products.

Injuries from Spraying or Seed Disinfection.—The successful use of chemical poisons for disease or pest control is based on the selection of compounds which will have the desired effect upon the pathogenes, inhibiting their growth or killing them outright, without causing serious injury to the crop plants which they parasitize. The principal preparations which have caused serious spray injury in agricultural practice are copper-containing fungicides, especially Bordeaux, lime sulphur or other sulphur sprays, arsenicals used for chewing insects and oil sprays used as contact insecticides. Of the many chemicals tried for disinfecting seeds, only a few have been widely used, the most important being mercuric chloride or corrosive sublimate, copper sulphate, or bluestone, and formaldehyde. Under certain conditions, all have caused injury, either by reducing vigor of growth or by reducing the actual germination percentage. Both spray injury and seed injury will be treated more in detail.

Injuries from Fumigation.—The use of fumigation as a method of distributing the chemical is also fraught with danger. The fumigation of potato tubers with formaldehyde for scab control resulted in so much injury that the method has never come into general use (Morse, 1907). Cyanide fumigation of greenhouses for the control of white flies or other insects frequently results disastrously, since different species of plants show a varying tolerance, and seedling plants are generally more sensitive than more mature plants. This makes it more difficult to be sure of a safe dosage, since frequently mixed cultures of varying ages must be protected. It has recently been shown (Butler and Jenkins, 1930) that only a neutral or nearly neutral Bordeaux should be used on plants to be cyanided. The injury when non-neutral Bordeaux has been used is due to the formation of cupric cyanide. Even in cyanide fumigation of citrus trees in the open, the dosage must be very carefully adjusted to avoid injury. In this connection, the practices of anæsthesia to advance the

date of blossoming may be mentioned. The fumes of ether, chloroform or other anæsthetic may have the desired stimulating effect, but they may also result in the death of the plant; hence the treatment requires special care.

Injuries Due to Soil Sterilization.—Chemical preparations may also be added to the soil to kill bacteria, fungi or insects. The persistence of these chemicals in the soil or their interactions in the soil may result in injurious after effects upon the crop to be protected or upon following crops. For instance, if sulphur is used in large amounts to control potato scab in contaminated soils (Sherbakoff, 1914), the yield of following crops is reduced. The use of cyanamid, as recommended by Watson (1917), for the sterilization of soil for eelworms or nematodes caused so much burning or scorching of crops planted after the treatment that it was necessary to modify the practice (Watson, 1921). Carbon bisulphide has been used for soil disinfection for nematodes or soil-infesting insects, but its use is difficult or impossible except in unoccupied areas, because of the poisonous effects upon the roots of plants.

Injuries Due to Refrigeration.—Brief mention may be made of the injurious effects of refrigeration in the storage or transport of fruit. The low temperatures are used to slow down the life processes in the fruit tissues and to retard the growth of rot-producing fungi. In wrapped peaches, the browning and death of external patches, known as "ice scald," illustrate one of the difficulties encountered in refrigeration (Hill, 1913). The internal browning of the Yellow Newtown apple is due in part to holding the fruit for a prolonged period at too low a temperature (Ballard *et al.*, 1922). Brown heart of apples has been shown to develop under those conditions of refrigeration which were designed to prolong the storage life by retarding scald and inhibiting the action of rot-producing fungi (Kidd and West, 1923).

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BORDEAUX INJURY

Through the work of the U. S. Department of Agriculture, Bordeaux mixture was introduced into this country in 1887. It soon came to be the generally accepted fungicide for the protection of growing crops from the attacks of various parasitic fungi. The principal complaints of injury from its use have been from orchardists. Apple and peach trees have been most generally affected. The injury from Bordeaux has been known under such names as Bordeaux scald, spray injury, Bordeaux burning, spray russeting, cork russeting and yellow leaf.

Types of Spray Injury.—The application of a chemical compound as a spray to the foliage or other aerial parts of crop plants may cause certain types of injury. Without specifying any particular fungicide, the injurious effects which may follow spraying may be grouped as follows: (1) *Leaf injuries:* staining, spotting, shot holing, burning, yellowing and defoliation or leaf fall. (2) *Twig injuries:* spotting, general discoloration, cankers, gummosis or dieback. (3) *Blossom injuries:* blighting of parts and failure to set fruit. (4) *Fruit injuries:* staining, spotting or russeting, malforming and cracking, burning, reduction in size, dropping or modification of composition. (5) *Entire-plant injuries:* general necrosis and death.

Not all the effects will follow from the use of a single fungicide on a given plant, but different crops will exhibit varying responses. It may be noted, however, that the possible injuries are very similar to the effects of the parasites which the sprays are intended to prevent or control.

History of Bordeaux Injury.—Early in the use of Bordeaux in this country, the injurious effects upon the apple were noted, and attention has repeatedly been called to the dangers of spraying various crops with copper preparations. Despite its injurious effects, Bordeaux continued to be the prevailing orchard fungicide until the discovery of lime sulphur, which has supplanted it in the control of many diseases. Even as early as 1889, Weed stated that Bordeaux was not safe for spraying apples for scab. There were numerous reports of a similar character in experiment-station literature in this country, and European investigations also were much concerned with the injurious effects of this spray. Some of the earlier reports in America were by Jones (1892), Green (1893), Beach (1894), Lodeman (1894–1896), Duggar (1898) and Stewart and Eustace (1902). The urgent need for a safe spray for the control of the brown rot of the peach led to the work of Bain (1902), a physiological investigation with special reference to the injurious effects of fungicides on peach foliage. Despite various recommendations for the prevention of injury in apple orchards, growers still experienced much trouble, and this led to the detailed investigations of the whole subject of Bordeaux injury by Hedrick (1907), who gave special attention to cause, favoring factors and methods of prevention. Mention should also be made of the work of Crandall (1909) on Bordeaux mixture in general and his study of the

relation of meteoric factors to foliage and fruit injury. Severe injury to peaches in New Jersey in the spring of 1909 led to a special study by Groth (1910) of Bordeaux injury on peaches.

Symptoms and Effects of Bordeaux Injury.—Since it is not the custom to spray fruit plants with Bordeaux when they are in bloom, injury to the blossoms has but little practical significance. Both foliage and fruit may be injured, and in the apple the fruit injuries have been the principal handicap to the use of Bordeaux.

Some of the common names of the injury, as "spray russeting" and "cork russeting," indicate in a very general way the nature of the injury on the fruit. Injured specimens always become more or less rough and russeted, and the layers of damaged cells thick and corky (Hedrick, 1907).

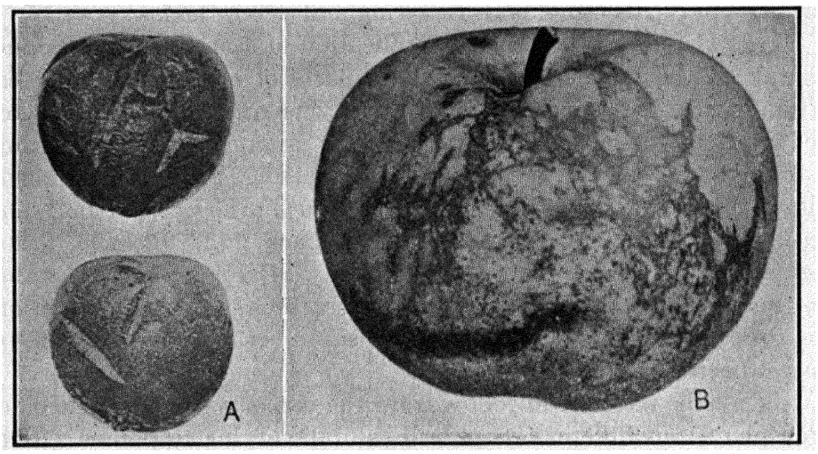

FIG. 68.—Bordeaux injury. *A*, severe Bordeaux injury of half-grown Baldwin apples; *B*, Bordeaux injury on a Rhode Island Greening apple. (*After Hedrick, N. Y. (Geneva) Exp. Sta. Bul.* **287.**)

The injury appears first as small dark or brown specks, less than 1 millimeter in diameter, and these are more or less isolated, or they may be so numerous as to coalesce and form rather extended russeted areas. The location of the injured areas will depend upon the position of the fruit at the time when the spray was applied, being on the surface to which the greatest quantity of the spray material adheres. Severe injuries to young fruits may cause more or less distortion in shape, due to localized atrophy or shrinking of tissue or in other cases to teat-like malformations. As the severely affected fruits grow older, deep cracks may form, and these may be healed over with the formation of cork cells. The appearance of minute red spots, centering at the lenticels, on yellow-skinned apples has been attributed to the effects of Bordeaux (Barss and Smart, 1921), although other agencies may also produce similar effects. Moderate russeting is of main concern as affecting the appearance of the fruit, but the market value may be ruined by the more severe types of

injury. Bordeaux-injured apples have poor keeping qualities, since they lose moisture more rapidly than normal fruits and are more easily invaded by rot-producing fungi. A very similar russeting of the fruit is caused by frosts during the young stages of growth (see Frost Injury, p. 160).

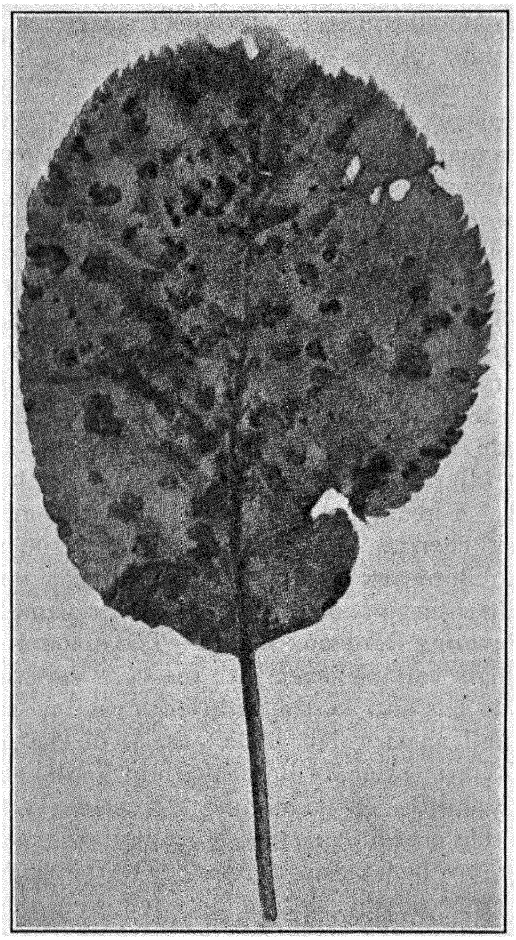

FIG. 69.—Bordeaux spotting of apple leaf. (*After Hedrick, N. Y.* (*Geneva*) *Exp. Sta. Bul.* **287**.)

In addition to the production of localized lesions, Bordeaux has been noted to cause reduction in the size of cherries and also an increased susceptibility to frost injury (Dutton and Wells, 1923).

On the foliage, Bordeaux injury very greatly resembles the leaf spot supposed to be caused by any one of several fungi. The affected leaves first show dead, brown spots; the majority of these spots are circular or roundish, with a diameter of 2 or 3 millimeters, but they are of various shapes and sizes; many are of irregular outline and so large as to appear to have been caused by the coalescing of smaller spots. The line of demarcation between the dead tissue of the spots

and the living green of the leaf is well marked, so that the spots are very conspicuous (Hedrick, 1907).

If the spots are few in number, no other effects may be noticed, but if numerous, the intervening tissues may turn pale green or yellow and the leaves fall. The amount of leaf fall varies from almost none to nearly complete defoliation. A loss of one-third to one-half of the foliage was considered a fair average for New York orchards in 1905, a season very conducive to Bordeaux injury. In the most extreme type of leaf injury, the affected trees may look as though they have been scorched by fire.

The stone fruits in general, and especially the peach, are more sensitive to Bordeaux or to other copper fungicides than the apple. Spotting, burning and defoliation will be more severe with the peach than with the apple when both are subjected to the same conditions, but the peach will also show a shot holing of the foliage. The shot-hole effect is not a response that is peculiar to Bordeaux injury but is simply a host response to localized injury of leaf tissue and may be induced by various other factors (Duggar, 1898). Even very weak Bordeaux may cause injury to peaches if certain favorable conditions prevail. In addition to the foliage and fruit injuries, a pronounced reddening of the sprayed twigs has been noted. A reduction in size of Morello and Montmorency cherries, or "small cherries," has been attributed to the use of Bordeaux (Dutton, 1928). Bordeaux spraying has also been noted as greatly increasing drought injury in ginseng culture (Wilson and Runnels, 1931).

Conditions Favoring Bordeaux Injury.—The amount of spray injury has been exceedingly variable, even with the same varieties and with the same formula. It has also varied with localities, one region reporting heavy damage and another but little. Some of the most important features which favor or promote Bordeaux injury are as follows: (1) the use of excessive quantities of the mixture; (2) the use of too strong solutions or of those that contain an excess of copper; (3) mechanical injuries to the foliage, due to the presence of fungi or to the work of insects; (4) damp, foggy or rainy weather immediately following the application of the spray. It has been the experience of growers that the more spray they apply the greater the injury; *i.e.*, a heavy dripping spray will cause more injury than a finely divided mist spray which covers the leaves and fruit with a thin film. With the thin films, there is never so high a concentration of copper at any one point as when drops collect and thick copper deposits are formed. As a general principle, it may be stated that increase in the copper sulphate content of Bordeaux will increase the amount of injury.

Early formulas were much stronger than those used later. In 1888, the U. S. Department of Agriculture formula was copper sulphate 6 pounds, lime 4 pounds, water 22 gallons; but by 1896 the 50-gallon formulas became the rule, and the 4-4-50 formula, or 4 pounds of copper sul-

phate, 4 pounds of lime and 50 gallons of water was one of the common standards, although the copper sulphate content has varied from 2 to 6 pounds and the amount of lime from 2 to 10 pounds. Experience showed that the old formulas contained more copper than was really necessary to give the desired protection and that the danger of injury was much greater. It has been possible to obtain good protection against scab of apples with a 3-3-50 formula, and for various troubles on cherries and more susceptible stone fruits a 2-4-50 formula has been reasonably safe, while in some regions a 3-10-50 formula has reduced the amount of russeting in apples.

Bordeaux mixture made by using equal quantities of copper sulphate and lime or an excess of lime does not prevent injury, but the danger of injury is somewhat lessened. Hedrick (1907) opposed this view, but his conclusion has not been upheld by more recent practices. The influence of insect injuries and the presence of fungous parasites on the foliage have been mentioned by various workers as increasing the amount of Bordeaux injury. This may be explained by the easier penetration of the copper through the abrasions or breaks in the leaf surface.

Many of the anomalies of Bordeaux injury can be explained by the pronounced influence of the weather conditions which prevail during and immediately following the spraying operations. The general opinion of fruit growers that wet weather favors the trouble was confirmed by experiments by Hedrick (1907). This relation of meteoric moisture to injury was investigated in more detail by Crandall (1909), who states:

> The importance of rain and dew as agents causing brown spotting of foliage following applications of Bordeaux mixture is well attested by the uniform results obtained from the experiments with covered and uncovered trees. Two trees were sprayed heavily; one was left exposed, the other was protected from all rain and dew. This was repeated during three seasons. In each year, the foliage of the exposed tree was more or less injured by brown spots, while the tree protected from rain remained free from injury.

The increased injury during humid, cloudy periods is now a generally accepted fact, but the theories as to the exact way in which the injury results have been somewhat at variance.

Etiology.—Bordeaux mixture is made by bringing together a solution of bluestone, or copper sulphate, and milk of lime made by slacking quicklime in water. Much has been written about the chemical composition and the physical properties of this preparation, but these features cannot be discussed at this point.

> One important character of Bordeaux mixture is agreed upon by all chemists; *viz.*, that all but a trace of the copper is in the form of an insoluble precipitate. The clear liquid above this precipitate, which always forms when the mixture stands, has no value as a fungicide, the precipitate containing all of the fungicidal properties (Hedrick, 1907).

It is this finely divided precipitate which is deposited upon the plant surfaces in spraying, and under atmospheric conditions the copper hydroxide is changed into copper carbonate. Under bright, sunny conditions dissolved copper does not penetrate the leaf tissues and cause injury, but under the conditions which prevail during humid cloudy weather more is brought into solution and more penetrates the plant tissues, hence the increased injury.

It has been stated by various investigators that substances secreted by the sprayed plant or by germinating spores of fungi furnish conditions for the solution of small amounts of copper. It should be noted that during bright weather transpiration is active, the stomata are frequently more or less closed and the intake of carbon dioxide is in excess of the outgo, or nearly in balance, while in humid, cloudy weather transpiration is checked, the stomata are open, photosynthesis with its consumption of carbon dioxide is less active but respiration with its production of carbon dioxide is still active. A consideration of the above facts led Groth (1910) to formulate the following theory of Bordeaux injury in his study of the spray injury of peaches.

> An excess of CO_2, evolved in the shade, passes into the water standing on the leaf during wet weather. CO_2 is dissolved, and the carbonated water thus formed dissolves some of the copper. The copper solution diffuses through the water film into the stomatic chamber and kills the cells with which it comes in contact.

Bordeaux injury is generally slow in developing. It begins after the rains supply the requisite moisture and may continue to develop for weeks or even months. In the young growing structures, either leaves or fruits, the epidermis has not yet become cutinized, and hence penetration of the poison is possible through these unprotected walls by osmotic transfer. It was formerly stated that the late spraying of apples was dangerous, but Hedrick (1907) has pointed out that "Bordeaux injury on fruit comes from early spraying, after the blossoms have dropped, and it is not probable that much damage is done after the hairs have been shed and the stomata changed into lenticels."

Susceptibility of Different Species and Varieties.—Some species of crop plants are so tolerant to copper that little or no injury results from the use of Bordeaux, even under conditions that would seem to be especially favorable. This is true for the potato, a crop which must be protected from late blight by the use of Bordeaux. Of the plants commonly sprayed for fungous diseases, the peach and the Japanese plum occupy the other extreme and

> . . . are so easily injured that it is seldom profitable to spray them with this compound, since a strength of spray which will control fungi will usually injure the foliage of these trees. In spraying practice, it is found that the apricot and the Japanese plum behave much as does the peach when sprayed with Bordeaux

mixture and that the Domestica plums, while not so easily injured, yet not infrequently show harmful effects on both fruit and foliage (Hedrick 1907).

Cherries are more comparable to the Domestica plums, but sweet cherries are more sensitive than sour varieties. The grape is also injured by copper sprays, and much has been written concerning the injury from and the protection afforded by various preparations. The apple, quince and pear are about equal in their tolerance to Bordeaux. Both pears and apples show considerable variation in the resistance of different varieties to copper. Hedrick groups pears into varieties injured badly and those injured but little, the Anjou belonging to the former and Bartlett and Winter Nelis to the latter. Apples show a much greater variation in susceptibility, and resistance seems to be a definite variety character. "Fruit and foliage do not always show the same degree of immunity; *i.e.*, a variety may be susceptible to the injury on the fruit and comparatively immune in the foliage or the reverse" (Hedrick, 1907). Over 150 varieties of apples were classified by Hedrick as to their immunity to Bordeaux injury and grouped in the following divisions: (1) no injury or very slight; (2) slight injury; (3) badly injured; and (4) very badly injured. Important commercial varieties are found in each group, but the classification shows that the Russian varieties and crabs are generally more subject to Bordeaux injury than other varieties, although some important Russian varieties are highly resistant.

Prevention.—The danger of injury from the use of Bordeaux has led to its abandonment as an orchard spray whenever it has been possible to find a satisfactory substitute. This has led to the use of lime sulphur or sulphur dust in certain regions. For certain diseases of apples—*e.g.*, bitter rot, blotch and Pacific Coast anthracnose—Bordeaux is still the most satisfactory fungicide. In case Bordeaux must be used for spraying apples, it will be impossible to prevent some injury because of the influence of climatic factors over which the grower has no control, but the effort should be made to cut the injury down to as slight an amount as possible. The following recommendations should be the guide in so far as possible: (1) Consider the resistance to Bordeaux injury, but select for planting varieties that are otherwise adapted to the environment; (2) reduce the formula to the one containing the least amount of copper sulphate that will give the desired protection, and use an excess of lime and a casein spreader; (3) practice moderation in spraying; *i.e.*, spray with a fine mist that will cover but not drip heavily; (4) spray as nearly as possible in dry weather, avoiding damp, foggy or rainy periods; (5) remember that early sprayings are the most dangerous, and give special attention to cutting down injuries at that time.

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LIME-SULPHUR INJURY

Lime sulphur was first introduced into orchard practice as a substitute for Bordeaux mixture and was heralded by its advocates as a preparation which would eliminate spray injury, especially in the apple orchards, but it was soon found to cause several different types of injury.

History.—Lime sulphur was first used on a commercial scale as a summer spray by Cordley in 1908, and its general adoption as an orchard spray followed very quickly in the Pacific Northwest and in the eastern United States. Serious injury to peaches was reported by Scott (1909), and a year later Wallace (1910) made a special study of lime-sulphur injury, giving special attention to apples and peaches. His work was concerned mainly with foliage injury, as fruit injury seemed to be rare under New York conditions. The success which attended the use of lime sulphur for apple scab led to its substitution for Bordeaux in the spraying of potatoes for late blight, and its injury to the potato was revealed by the tests first published by Stewart and French (1912) and continued by Munn (1912, 1915). The introduction of lime-sulphur spraying into the hot irrigated valleys of the Pacific Northwest soon showed that the spray behaved differently under the prevailing climatic conditions. A new phase of lime-sulphur injury, the burning of the fruit, formed the subject of a special investigation by Safro (1913) in Oregon. The unsatisfactory character of lime sulphur as an apple spray led to the adoption of iron sulphide in the Pajaro Valley of California (1914). Neither this new formula nor lime sulphur proved satisfactory for powdery-mildew control in the Pacific Northwest, especially in the hot irrigated valleys, because of severe fruit injury or "sulphur sun scald."

Another and more severe type of injury was first reported from Nova Scotia in 1914, growers claiming that they had "sprayed their apples off the trees." This indirect injury to the fruit has been investigated in some detail by Sanders (1922), and a somewhat similar effect of lime-sulphur injury has been noted in other regions having about equivalent amounts of sunshine during the growing season.

Symptoms and Effects of Lime-sulphur Injury.—Lime sulphur may cause localized injury to either foliage or fruit, causing characteristic lesions. It may also cause the dropping of fruit as the result of interference with the physiological processes carried out by the foliage and in certain plants may have a retarding or inhibiting effect upon growth and cause reduction in yield without the production of evident lesions.

Foliage injury of the apple may be of several types.

Perhaps the most common type is the dull-brown spotting or marginal and tip burning which occurs where hanging drops of the solution have gradually become more concentrated during the drying process (Wallace, 1910).

In the case of lesions removed from the margin of the leaf, a scab infection or an insect injury usually marks their centers. With numerous scab infections the burning may be general and severe. Even in the absence of scab, heavy drenching of the foliage may result in the burning of large areas or of entire leaves.

In general, lime-sulphur solution, unless applied very weak, is likely to cause considerable burning of peach foliage. The occurrence and the character are quite different from those on apple foliage. In the latter case, the dark-brown spots or burned areas at the tip or margin of the leaf appear within about 2 days after the application. On the peach, it may be almost a week before the spotting is noticeable. Then, certain definitely outlined spots appear, usually rather pale green, with darker green or reddish-brown borders. In mild cases, it somewhat resembles the effect of the leaf-spot fungus; and, as in leaf spot, the injured parts finally drop out, leaving the shot-hole effect. "Very slight injury is sufficient to cause the falling of peach leaves, so that defoliation in severe cases is likely to be very noticeable" (Wallace, 1910). Under certain conditions, even dormant spraying with lime sulphur may cause injury. Attention has been called to the injury to peach twigs and buds in California when sprayed immediately after a severe north wind of 1 or 2 days' duration (Urbahns, 1931).

Experience in general has substantiated the conclusions of Wallace (1910) that:

If any russeting has been caused by lime sulphur, it is so little more evident than the natural russeting that has occurred quite commonly this season that it is very hard to distinguish between the two.

According to Bonns (1911), "Results from many experiments show absence or reduction of fruit russeting with the use of lime sulphur," but

more recently rather general russeting from the use of lime sulphur has been noted (Young and Walton, 1925). If used during periods of high temperatures and intense sunshine, it may cause a burning of the fruit, which may be called "sulphur sun scald," the lesions being very similar to those due to sun scald alone. The effect on the fruit is the appearance of a pale-brown, more or less circular area on the sun-exposed cheek of the fruit. As a result of the death of underlying cells, the spot becomes darker and flattened or even slightly depressed, and the affected area may be somewhat checked or cracked. This type of fruit injury seems to be confined very largely to the semiarid irrigated fruit districts, which are characterized by high summer temperatures and intense sunshine, or to exceptionally hot and dry seasons in regions which are normally fairly humid.

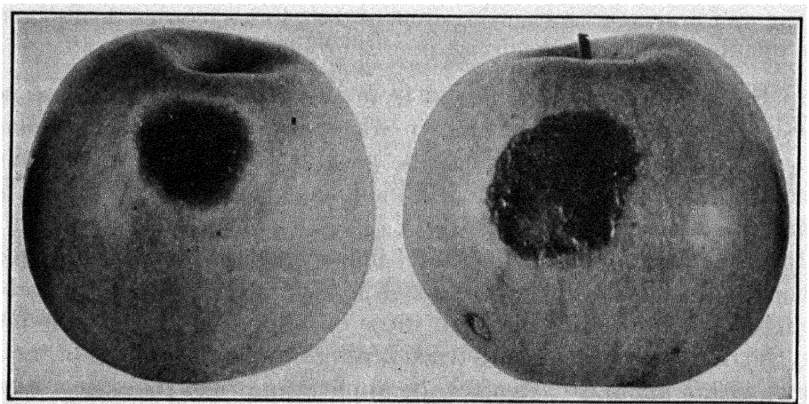

FIG. 70.—Sulphur sun scald of apples.

Until quite recently, this direct fruit injury was believed to be the principal danger to the fruit resulting from lime-sulphur applications, but the dropping of the fruit following the use of lime sulphur has caused serious losses under very different climatic conditions. A specific illustration given by Sanders (1922) may be cited from results obtained in Nova Scotia. Trees were sprayed with Bordeaux (3-10-50) and lime sulphur (1-40) each year for 7 years from 1915 to 1921, with the result that Bordeaux-sprayed trees produced an average of 341.2 apples per tree, while the lime-sulphur-sprayed trees yielded an average of only 73.25 apples per tree. It was also shown that the injury which caused the fruit to fall also reduced the size of those which did grow to maturity. This would indicate a checking or an inhibition of the photosynthetic processes as a result of the spraying.

The losses possible from lime-sulphur spraying may be judged from the analysis by Sanders (1922) of conditions in Nova Scotia:

In 1911, the Annapolis Valley produced approximately 1,700,000 barrels of apples. In 1912, the growers started using lime sulphur generally and continued

its use until 1919. During the 7-year period, the crop did not in any year reach 1,000,000 barrels. In 1919, the growers there changed to a 3-10-50 Bordeaux and used an appreciable amount of dusting material, with the result that they produced over 1,000,000 barrels. In 1920, lime sulphur was completely abandoned and a greater amount of dust used than ever before, and that year a crop of nearly 1,200,000 barrels resulted. In 1921, two-thirds of the orchards there were dusted and the remainder sprayed with the modified Bordeaux, and a crop of over 1,500,000 barrels was produced. In other words, changing to dusting and modified Bordeaux and the abandoning of lime sulphur have not only almost doubled the Nova Scotia apple crop, but it has given them three large crops of clean fruit in succession.

It was estimated that the yearly loss in Nova Scotia from 1912 to 1918 from the use of lime sulphur amounted to over 700,000 barrels. The dropping of the fruit has also been noted in a number of localities when lime sulphur is used but the earlier applications omitted. Since the dropping of the fruit would have been prevented by gradually accustoming the trees to lime sulphur by the earlier applications, the behavior noted is referred to as "sulphur shock."

The best illustration of the retarding or inhibiting effect of lime sulphur on a crop of a different character is shown by the comparison of Bordeaux and lime sulphur as a spray for potatoes. From tests carried out in New York during four successive seasons (1911–1914), Munn (1915) states that lime sulphur "aggravated tip burn, dwarfed the plants, shortened the period of growth and reduced the yield." The lime-sulphur-sprayed plants died 10 days to 2 weeks earlier than those in unsprayed rows, and the average reduction in yield per acre amounted to 28.5 bushels. Not only does lime sulphur have this inhibiting effect upon the life processes of the potato, but it is much less valuable than Bordeaux in the control of late blight. It has been stated that lime sulphur has a depressing effect when used as a spray for raspberries in the Puget Sound country, but this does not seem to be the case in recent Wisconsin tests.

Mention should be made of the relation of lime-sulphur spraying to the production of "springers," or swelled cans (Stevenson, 1926; Culpepper and Moon, 1929). These are caused by the formation of hydrogen and hydrogen sulphide, while the fruit may have a sulphide flavor, and the tins show sulphide staining.

Etiology.—Lime sulphur, which is made by boiling together lime, sulphur and water, contains calcium polysulphides (CaS_4 and CaS_5) and calcium thiosulphate (CaS_2O_3) as its most important ingredients. Both are soluble in water, but the former have been shown to be the cause of most of the injury. The other normal ingredients are practically harmless. Lime-sulphur injury appears a few days after the spray is applied, as opposed to Bordeaux injury, which is generally much delayed. This behavior, according to Wallace (1910), appears to be due to the fact that the polysulphides remain in the soluble form but a short time.

Lime-sulphur injury will vary more or less under constant climatic conditions, being influenced by condition and susceptibility of the plants sprayed, the concentration of the mixture, the kind of arsenical employed and the time and method of application, but climatic factors are of direct bearing, especially in the case of sulphur sun scald and the dropping of the fruit.

It is generally agreed that the intensity of injury from lime sulphur depends on the percentage of soluble polysulphides present. Lime sulphur is diluted on the basis of the Baumé test, but the specific-gravity test is an inexact basis for determining the polysulphide strength of the spray.

A dozen samples of lime sulphur all having the same specific gravity may show no two samples alike in per cent of soluble sulphides. Furthermore, a sample having a low specific gravity may have a greater per cent of soluble sulphides than a sample having a higher specific gravity (Safro, 1913).

Specific gravity is determined by all ingredients in solution; hence the density is no accurate measure of the power of a given solution to cause injury. Wallace believed that lime-sulphur injury was due solely to the direct action of the soluble polysulphides, but the experiments of Safro (1913) and others indicate that injury may result after the spray is dry. It seems to be true that injury at high temperatures is due to the rapid oxidation of the sulphur and the production of either sulphurous or sulphuric acid.

Safro (1913) attempts to account for lime-sulphur sun scald of apples on a physical basis rather than from chemical reactions. He states that the difference in the injury to sprayed and unsprayed fruit may be accounted for by the difference in the absorption of heat. The residue of lime sulphur deposited upon the fruit retards radiation and increases the absorption of heat, depending on its thickness, and hence sprayed fruit burns more than unsprayed because the tissues become more heated. This undoubtedly plays a part in the production of injury but can hardly be accepted as the sole cause. Whatever the explanation, it is true that sun scald is more severe in sprayed than unsprayed trees, and when the temperatures are high (95°F. or above) this holds true with lime sulphur, iron sulphide or elemental sulphur.

A very reasonable explanation has been given by Sanders (1922) for the dropping of apples and the injury to such crops as the potato, grape and other especially sensitive plants. The lime sulphur penetrates the stomata of the leaf surfaces and acts directly on the chlorophyll bodies, causing a discoloration that can be detected by microscopic examinations. This derangement of the chlorophyll apparatus inhibits or retards the photosynthetic process, and hence the young apples are "starved off the trees." This manner of action was further substantiated by certain tests:

(1) Lime sulphur applied to apples alone caused no fall; (2) when sprayed on the upper side of the leaves, there was likewise no dropping; but (3) when the undersides of the leaves were sprayed, most of the apples dropped off in the same manner as in "June drop." In this connection, it should be noted that stomata are present only on the undersurface of apple leaves. It is of interest to note that the foliage of the potato, grape and other plants which have stomata on the upper surface cannot be sprayed with lime sulphur without causing serious injury. This interference with the manufacture of carbohydrates will not only explain the dropping of the fruit, but it will explain the reduced size of that which does remain and also the reduced yields in a crop like the potato, which depends on the storage of carbohydrate food. It is suggested that the difference in the susceptibility of varieties to lime-sulphur injury is due in large part to the difference in the permeability of the leaf surfaces.

According to Sanders (1922):

> It was found that the intensity of the injury seemed to vary with the amount of sunlight during May, June and July. In England, New Zealand, the Kootenay valleys in British Columbia and in Nova Scotia fruit removal by lime sulphur seems to occur every year. In such areas as Ontario, New York State, New England, etc., where there is more sunlight, say an average of over 250 hours of sunshine per month during May, June and July, it would seem that serious fruit removal by lime sulphur occurs only in seasons when the amount of sunshine per month drops below that figure. Since chlorophyll depends on sunlight not only for its action in converting carbon dioxide into sugar but for its own actual formation, it can readily be seen that in years of plenty of sunlight the chlorophyll would be replaced almost as fast as it was injured, and the injurious effects on the crops and tree rendered almost negligible, whereas in years of little sunlight the injury might be severe in the same areas.

Prevention of Lime-sulphur Injury.—Since lime-sulphur injury is dependent in part on climatic factors and upon other features which are difficult to control, there is no certain method of completely eliminating injury. The following possibilities should serve as a guide: (1) Discard lime sulphur entirely for certain sensitive crops or for the more resistant crops under climatic conditions that are especially conducive to injury. The substitute must vary with the crop, the temperatures which prevail, the amount of sunshine and the pests to be controlled. (2) For tolerant crops, reduce the concentration to the lowest point which will give the desired protection. For the apple, 1-30 of the 33° Bé. concentrate is reasonably safe, but 1-50 is as strong as should ever be used for summer applications on the peach, while some recommendations call for nothing stronger than 1-100. So much injury with even this strength is likely, that such fungicides as self-boiled lime sulphur or dry-mix lime sulphur are recommended as substitutes. (3) Use arsenate of lead as the arsenical, since arsenite of lime, arsenite soda and Paris green are likely to cause

serious foliage injury when mixed with lime sulphur. Danger of injury with the combined spray of lime sulphur and arsenate of lead is materially lessened by the use of a casein spreader (Thatcher and Streeter, 1924). Injury is lessened by the use of ½ to 3½ pounds of ferrous sulphate per 50 gallons (Dutton, 1928) or in the case of the New Jersey dry-mix by the addition of 3 to 4 pounds of ferric oxide per 50 gallons (Ginsburg, 1927). (4) Spray with moderation, since overdrenching is likely to cause injury, as in spraying with Bordeaux. It has been claimed that the use of the spray gun in the orchard has increased injury, presumably by covering the lower leaf surfaces to a greater extent than by the old method with mist nozzles and extension rods.

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INJURY FROM OTHER SPRAYS

Brief mention may be made of injury from some of the other spray mixtures, especially arsenicals and coal oil or its products. Of the three most important arsenicals, London purple, Paris green and lead arsenate, the first is the most injurious, Paris green less dangerous and lead arsenate the safest. The injury from an arsenical is due to the original content of water-soluble arsenic or to interactions in a mixture which liberates free arsenic. London purple was largely discarded in favor of Paris green, because of its large amount of soluble arsenic, while Paris green, which has shown a varying percentage of free or water-soluble arsenious oxide (Colby, 1903), has been very largely supplanted by lead arsenate, which contains but very little free arsenic. Recent studies (Swingle, 1929) have shown that both arsenious and arsenic acid are present in commercial lead arsenate and that at low concentrations both are about equally toxic to peach foliage when compared on the basis of metallic arsenic content. At higher concentrations, however, arsenic acid is more toxic, possibly because of its greater penetrating power. The author concludes that it is "impossible to reduce the soluble arsenic in acid lead arsenate sufficiently to prevent serious injury when used on tender foliage." For safety on tender foliage, material must be added to neutralize the free arsenic. Injury has been lessened by the addition of hydrated lime (Campbell, 1926). In wet seasons, this has been nearly worthless on peaches, but under these conditions very satisfactory results have been obtained with 4 pounds of zinc sulphate and 4 of hydrated lime added to 1–50 lead arsenate (Hurt, 1931).

Lead Arsenate Injury.—Spotting or burning of foliage may result from the use of lead arsenate, although this rarely causes severe injury except on the more sensitive species. Several features of interest in the investigations of Fernald and Bourne (1922) may be noted: (1) Neutral lead arsenate was generally the least injurious; (2) clear-weather spraying was safer than cloudy; (3) spraying at high temperatures is safe if the humidity is low; (4) spraying at high humidities is safe if the temperature is low. "From the evidence at hand it would seem that, with reliable arsenicals properly made, mixed and applied, injury results from the combination of temperature, humidity and light factors." The dwarfing, shriveling and dropping of English Morello cherries has been shown (Gloyer, 1926) to result from pedicel injury from acid lead arsenate, when used either alone or in combination with Bordeaux, lime sulphur or sulphur dust. Similar injury was recorded on prunes.

Exceptional injury has resulted from lead arsenate spraying in numerous cases in which growers have used lye instead of a safe spreader. The foliage injury has simulated the leaf spot resulting from the black-rot fungus so closely that it has been mistaken for that disease in a number of cases. The addition of the lye liberated free arsenic, which caused the injury. Under the same treatment, the fruit showed numerous minute black-scorch specks.

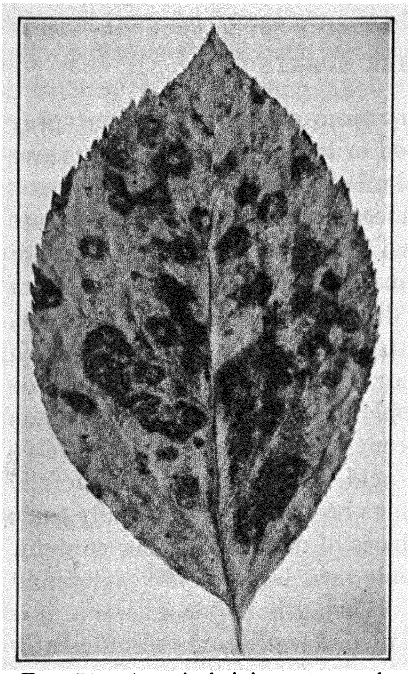

FIG. 71.—Arsenical injury to apple leaf.

An interesting type of injury in citrus fruits has recently been investigated by Gray and Ryan (1921). Oranges sprayed with "Tizit" and "Victory," proprietary preparations containing soap powder, sulphur and lead arsenate, had their acidity reduced to "roughly 50 per cent of the normal acidity on fully ripe oranges," but in many cases, especially Valencias which hung on the trees beyond the usual harvest time, the reduction was even greater. It was their conclusion that the slow liberation of a soluble form of arsenic was the cause of the reduced acidity, and they demonstrated that the changed acidity resulted when acid lead arsenate was used but did not appear when a basic lead arsenate was employed.

Consideration should be given at this point to two reports by Headden (1908, 1910) on the "arsenical poisoning of fruit trees." Three forms of arsenic poisoning were recognized: (1) *systemic arsenical poisoning*, due to the distribution of arsenic throughout the tree, resulting in a disturbed nutrition and growth and sometimes ending in death; (2) *corrosive arsenical poisoning*, due to localized attacks, affecting the tree at the crown, below the surface of the soil and frequently the large roots also, causing death and disintegration of the bark and cambium and internal discolorations of the wood; (3) a *bleeding*, due to the combined action of lime and arsenic. There are some weak points in the arguments and conclusions of Headden, and Balls (1909–1910) attributes the similar condition of trees in Utah to alkali and seepage water, while Grossenbacher (1909, 1912), in studies carried out in New York, attributed the crown rot to unfavorable winter conditions or winter injury. There are positive cases of crown corrosion due to alkali in our western irrigated semiarid lands, and the collar- or crown-rot type of winter injury is not uncommon.

From the more recent work of Swingle and Morris (1917), some additional evidence is presented that arsenic poisoning may play a part. They state that: "We have established conclusively that arsenical compounds used as insecticides can be made to injure the crowns of trees under conditions very similar to those that result from some orchard practices." It still remains an open question as to the exact part played by winter conditions, alkali and soluble arsenic in the injury and death of fruit trees. It is conceivable that all three factors may be operating under certain conditions, while in others only one or two of these injurious forces may be affecting the injured trees.

During the last few years, the requirement that market fruit, especially apples and pears, must comply with the Federal standard for freedom from arsenical spray residue has focused attention upon the whole problem of lead arsenate and its use as an insecticide. Two aspects are involved: (1) the cleaning of fruit at harvest time to reduce the arsenical residue to the quantity permitted; (2) handling the fruit in such a way as to prevent arsenical calyx burning during the cleaning process. Washing apples or pears in either acid or alkali cleaners is now necessary, except when conditions have required but few applications of lead arsenate. Calyx burning from free arsenic was known to occur in the orchard previous to harvest, before the days of spray-residue removal, but under certain conditions arsenical burning has resulted in severe form as a result of the cleaning. This is now largely prevented by the use of improved machines, the proper rinsing of the fruit to remove the free arsenic or the use of a neutralizing rinse. Under field conditions, heavily sprayed fruit has shown considerable calyx burning following late fall rains or if allowed to stand in the picking boxes in the orchard during rain periods. One of the serious aspects of arsenical calyx burning is that it offers an avenue for the entrance of blue mold or other decay-producing fungi.

Injury from Contact Insecticides.—Before the days of Black-leaf 40, when kerosene and kerosene emulsions were the common contact insecticides, there were numerous reports of severe burning or injury, especially to vegetative structures, but the use of the tobacco preparations has largely removed this danger. The use of distillates in the spraying of citrus fruits (Volck, 1903) has resulted in the spotting and yellowing of foliage with more or less defoliation and in the spotting and dropping of fruit. The physical basis of the injury is supposed to be from the insulation or sealing over of parts with the consequent interference with the normal gaseous exchanges, while the chemical basis of the injury is the absorption of volatile products.

During recent years with the introduction of oil sprays for the protection of trees during their dormant period, cases of very severe injury have been reported, when very low temperatures followed the application of

the spray. Whole blocks of trees have been killed outright, and as a result of these experiences the use of an oil spray during the winter when there is danger of zero weather is considered unsafe. No adequate explanation of this injury has been offered, but it seems possible that when low temperatures prevail, the sealing and insulation of the entire aerial structure by a coating of oil may so affect the internal oxygen-carbon-dioxide ratio as to kill the protoplasm, while at higher temperatures the critical ratio would not be reached. This idea is suggested from the recent studies of brown heart of apples under refrigeration (see p. 138). Lesser degrees of injury from oil sprays have been noted from dormant applications including twig and bud killing. Under certain conditions when dormant applications have caused no apparent injury, delayed dormant applications have killed blossom buds or delayed flowering.

The rather extensive use of summer applications of oil in the control of scale, red spider, leaf hopper and codling moth have revealed many cases of rather pronounced injury to both citrus and deciduous-leaved fruits. These injuries include yellowing, stunting, spotting, burning and abscission of foliage and dwarfing, spotting, russeting, dropping, poor coloration, scalding and delayed ripening of fruits. In certain cases, there has been a complete dropping of stone fruits soon after an oil spray early in the season, the fruit being "sprayed off" the trees.

Light oils are much less likely to cause injury than heavy oils of high viscosity. It has been shown that they penetrate the leaves mostly through stomata and are translocated to adjacent tissues. The injurious effects are thought to be physical rather than chemical, including interference with transpiration, respiration and photosynthesis. There seems to be a lack of unanimity in current publications as to the exact factors involved in the production of injury. In general, spraying at times of high humidity or at times of high temperature has given the most injury.

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INJURY FROM SEED DISINFECTION

Many parasites of crop plants are seed borne, being carried either in or on the seed. The smuts of cereals have become so widespread that treatment of seed grain to kill the parasite has become general in many sections of the country. Hot-water treatments have been used, especially for the loose smuts of wheat and barley, diseases in which the smut fungus is an intraseminal parasite, being present as a dormant mycelium. For the cereal troubles in which the organism is carried as spores on the surface of the seed, some chemical agent is generally used, and the seed is steeped, sprinkled or sprayed with the fungicide or coated with the poison in the form of a very fine dust.

Seed Injury from Hot Water.—The modified hot-water treatment of wheat has been noted to cause more or less injury when the seed is subjected to the action of the hot water for a sufficient length of time to kill the internal mycelium of the loose-smut fungus (10 minutes at 54°C.; allowable range 52, to 55°C.). The seed injury from this treatment has recently been studied by Tapke (1924), and the following effects were noted: (1) a reduction in the germination percentage; (2) an abnormal germination as indicated by small spindling seedlings; (3) a retardation in the rate of emergence of seedlings, and a slower growth during the seedling stage; (4) fewer culms per plant and possibly decreased yields. The average germination percentage of 33 varieties was 87.6, while the same varieties when treated by the standard hot-water method gave an average germination of only 52.7 per cent. It was further shown that this injury was due to the physical conditions of the seed coats; as treated, hand-threshed seed with no breaks or cracks in the seed coats gave a germination practically equal to untreated grain. The seed-coat damage is due largely to machine threshing and varies with the variety, the dryness at the time of threshing and the speed of the cylinder. Reduction in germination occurs when the seed-coat injuries are over the endosperm, but it is more pronounced when they are over the embryo. The amount of injury will vary during different seasons; hence the reduction in germination from the hot-water treatment cannot be definitely predicted but must be determined for each lot of seed. Any method of threshing which will reduce seed injury will lessen the reduction in germination following the hot-water treatment.

Seed Injury from Copper Sulphate.—Although copper sulphate has been used as a standard seed disinfectant for many years, especially in the control of cereal smuts, it has long been known that its use at strengths effective for the control of smut results in injurious effects to the treated seed. The strength of the solution and the time of treatment have been varied, pretreatment and aftertreatment practices tried out and various substitute fungicides introduced, in the attempt to reduce the injury to the lowest possible figure.

The injury from copper sulphate treatment has generally been measured in terms of the reduction in the percentage of viable seed, which may frequently show a drop from 90 to 100 per cent germination of untreated wheat to 35 to 60 per cent germination when given the standard bluestone treatment (1 pound to 5 gallons water for 5 to 10 minutes). It has been shown that the toxic action of the copper also causes a pronounced retardation of growth when the treated seed is planted in the field and that many seedlings which do grow make an abnormal development, with curved, deformed plumule and poor root growth. The injured seedlings also fall an easier prey to injurious soil fungi. Those most severely injured are never able to emerge from the soil, while others less injured may recover somewhat and later become nearly normal.

As early as 1872, Nobbe discovered that the percentage of germination of wheat depended largely on the extent to which the seed coats were broken or cracked, and this protective action of the seed coats has been rediscovered or confirmed by numerous workers since that time. The seed injury in fungicidal treatments is the result of the penetration of the copper sulphate through the breaks or cracks, so that it acts directly on the embryo seedling. Experience has shown that oats are more susceptible to copper sulphate injury than either wheat or barley. Since the pioneer work of Kühn in 1872, an after treatment of vitrioled grain has been recommended to eliminate the injury or to reduce it to a safe amount. Immersion in milk of lime (1 pound lime to 10 gallons water) has been commonly recommended for wheat, while some instructions have called for the dusting of the treated grain with air-slacked lime. This was recommended in a French method of treating oats. Since the introduction of formaldehyde, the bluestone formulas for the treatment of oats have been largely discarded, and their use for wheat has been continued only in those regions in which a soil contamination is an important source of the parasite (see Bunt of Wheat). Wheat sowed immediately after treating with copper sulphate shows the most injury, but according to Neuweiler (1928) complete drying for 28 days resulted in no loss of germination.

Formaldehyde was discovered by a German chemist, Hoffman, in 1867 and was first used in America by Bolley in 1893 in North Dakota for the treatment of the seed of cereals (Bolley, 1897). It came into use very rapidly, one of the principal recommendations being the negligible reduction in germination following its use at effective strengths. Use under diverse conditions soon showed that seed treated with formaldehyde might suffer just as severely at times as when treated with bluestone.

Seed Injury from Formaldehyde.—While it was admitted that formaldehyde treatment of cereals caused some reduction in germination of the seed, one of the earliest demonstrations of serious injury was afforded by the work of McAlpine (1906), which showed the danger of

allowing the seed to dry after treatment. Since that time, essentially similar results have been obtained by numerous workers, and many illustrations from field experience in the dry-farming districts have shown the danger of seeding formaldehyde-treated wheat in the dust. In certain cases, such heavy losses have been experienced that reseeding has been necessary. In carefully controlled germination tests, it has been shown that planting formaldehyde-treated seed in dry soil in which germination was delayed for 3 days in many cases doubled the seed injury over that occurring when the seed was planted in moist soil which permitted immediate growth (Zundel, 1921).

It was first suggested by Darnell-Smith and Carne (1914) that storage injury of the treated seed was due to the formation of a solid condensation product, or polymer, of formaldehyde, and they showed that washing the seed immediately after treatment prevented injury by removing this deposit. It remained for Müller and Molz (1914) to prove that this polymer, paraformaldehyde, is very injurious to wheat when mixed with the soil. This behavior of formaldehyde on the desiccation of the treated seed is now held to be responsible for many of the conflicting reports as to the injury resulting after the use of the standard formula. This phase of formaldehyde seed injury has been studied recently by Miss Hurd (1920), and according to her conclusions:

> The solid paraformaldehyde, being volatile, is constantly breaking down into formaldehyde gas. This gas, being thus concentrated and held so close to the seed, penetrates it slowly, going into solution in the testa. . . . The degree of post-treatment injury depended primarily on atmospheric humidity during the storage period. In atmospheres damper than 70 per cent humidity, the treated seed can be kept indefinitely without ill effects. In those of 70 per cent and less, there is decided injury, which is most severe in the intermediate humidities, gradually decreasing in the lower ones.

In studies of copper sulphate injury, it has been shown that unbroken seed coats are an almost perfect protection against reduction in germination percentage and abnormal growth. This does not hold true to the same extent for formaldehyde, but it has been shown that the unbroken seed coats are a partial protection against injury, affording "absolute protection against short exposures to strong formaldehyde solutions and partial protection against post-treatment injury" (Hurd, 1921). Recent studies by Atwood (1922) have confirmed the entry of formaldehyde into wheat through the seed coats and have shown that its absorption slows down the normal physiological processes, some demonstrated effects being retardation of diastatic activity and a weakened respiration. The degree of injury is affected by the temperature at which germination occurs, being most pronounced at low temperatures (Gassner, 1926).

A consideration of the way in which formaldehyde causes injury to wheat will show that in some regions its use will be likely to cause much

seed injury, while in others it may be used with reasonable safety. The predisposing factors in the semiarid or dry farming regions are: (1) the large number of broken or cracked seed as a result of harvesting and threshing during a period of extremely dry weather; (2) the common prevalence of medium air humidity at the time when seed is being prepared for seeding; and (3) the need for and the practice, in some sections, of seeding in the dust rather than waiting for a rain. Formaldehyde injury to wheat has therefore been most pronounced in fall-seeded crops in the arid regions and a minor factor in spring seedings.

Seed treatment by formaldehyde gas with the gas grain treater has not proved satisfactory, as it has not afforded uniform disinfection throughout the sack and has caused very pronounced reduction in germination (Kienholz and Smith, 1930).

The Prevention of Injury from Bluestone or Formaldehyde.—It is possible to avoid seed injury entirely by substituting some of the newer fungicides for the old bluestone and formaldehyde methods. Copper carbonate dust and several organic mercury compounds not only cause no seed injury but have a stimulating effect upon physiological processes and consequently induce a more rapid germination and a more vigorous growth.

If either bluestone or formaldehyde must be used, the amount of injury can be greatly reduced by observing the following precautions: (1) Avoid too strong solutions or too long steeps; *i.e.*, follow standard recommendations; (2) do not attempt to dry formaldehyde-treated grain and hold it before seeding, but treat the grain so that it can be seeded while still moist; (3) remember that wet grain will start to germinate if temperatures are favorable and may suffer injury from heating or molding previous to seeding; (4) do not plant formaldehyde-treated grain in the dust; (5) bluestone-treated seed may be planted in the dust with safety if it has been properly treated and may be dried before seeding without increase of injury (some workers even claim improved germination from complete drying); (6) protect wet grain from freezing.

In addition to the above precautions, two direct methods of reducing seed injury are available: (1) presoaking and (2) an after treatment. Presoaking wheat not only eliminates seed injury due to the use of either formaldehyde or bluestone but also increases germicidal efficiency. The method as tested and recommended by Braun (1920) consists of soaking the seed in water for 10 minutes and covering for 6 hours previous to treating. This is also effective for barley, oats and corn and with certain variations should give results with any crop for which either the formaldehyde or the bluestone treatment is necessary. In using the presoak method, it is recommended that the time of soaking should be long enough for the seeds to absorb about 30 per cent of their weight of water. The physiological basis of the efficiency of the presoak method is the reduced

diffusion of the poison into the seed during the period of treatment so that its concentration does not reach the danger point. Braun (1922) has also shown that formaldehyde-treated seed which has been given the presoak treatment may be held for several days or a week before planting without appreciable injury to germination.

An afterbath in milk of lime (1 pound quicklime to 10 gallons water), following the steeping of wheat in bluestone, has long been recommended as a means of reducing the seed injury, and more recently (Zundel, 1921) it has been shown that this same treatment is effective when formaldehyde has been used. This protective action of the lime bath in the case of formaldehyde-treated seed is not due to the lime, but the same results can be accomplished by soaking in water alone, as was first shown by Darnell-Smith and Carne (1914) and since rediscovered by other workers.

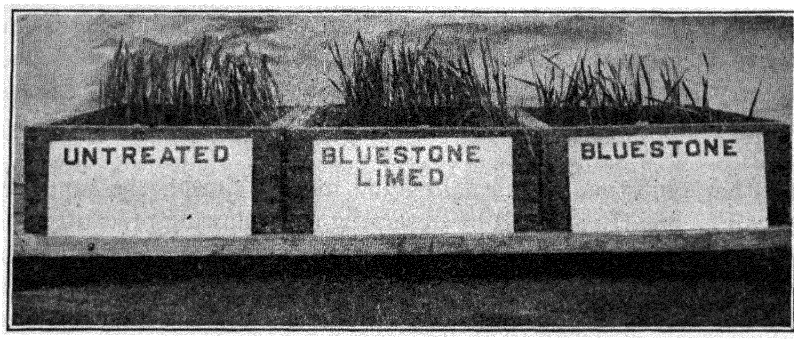

FIG. 72.—Germination injury to wheat treated with bluestone. (*Photograph by G. L. Zundel.*)

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SECTION III

VIRUS AND RELATED DISEASES

CHAPTER XII

GENERAL NATURE OF VIRUSES AND TYPES OF VIROSES

The virus diseases of plants constitute one of the great groups of plant troubles which stand out rather distinctly from those due to non-parasitic or environmental factors as well as from those in which a visible parasite is the causal agent. The virus troubles with their varied symptomology and effects have one feature in common, *viz.*, they may be transmitted from diseased to healthy plants by an infectious principle—the virus or the so-called *contagium vivum fluidum* the exact nature of which is unknown. Certain conditions of common occurrence in plants, like variegation or non-infectious chlorosis, seem to be very closely related to the virus troubles and may well be considered at the beginning.

Non-infectious Chlorosis.—In non-infectious chlorosis, there may be a more or less uniform yellowing of the foliage, or the leaves may show areas of white or yellow mingled with regions of normal green, giving the type of chlorosis that is called "variegation." Several different types of variegation may be recognized: (1) marginal variegation, with narrow or broad zones of pale tissue marking the edge of leaves or leaflets; (2) sectional variegation, with the yellow and green areas distributed over the leaves and stems or leaves only, in the form of blotches, spots, bands or stripes; and (3) marbled and pulverulent variegation.

When genuine chlorosis or variegation appears in food plants, it is considered to be a detrimental or diseased condition, since the photosynthetic power of the plant is modified or reduced in accordance with the degree or intensity of the yellowing (Schertz, 1921). When, however, these peculiarities of the foliage appear on ornamental plants, they are considered as adding to their decorative value, and the variegated forms are preserved. These abnormal or chlorotic forms appear in the trade as horticultural varieties of the normal species or variety from which they originated, under such names as *aurea*, *variegata*, *alba*, *argentea*, *aureo-*, *albo-* or *argenteo-marginatis*, etc. This type of chlorosis may be illustrated among herbaceous forms by ribbon grass (Phalaris), zebra grass (Miscanthus), variegated periwinkle (Vinca) and variegated varieties of the common garden nasturtium; among shrubs by the golden elderberry, a form with golden-yellow or yellowish-green leaves, and many variegated forms such as the variegated elderberry (*Sambucus spp.*), burning bush (Euonymus) and althea (Hibiscus); and among trees, by variegated holly

(Ilex), the golden-leaved English elm, the variegated maples and the golden box elder.

Variegation may appear spontaneously in almost any herbaceous or woody species either growing wild or under cultivation, without the operation of any known inciting factors. In food plants, these variegations are either ignored or eliminated by selection; while in ornamental plants, they may be propagated. In the great majority of cases, variegation can be propagated only by the use of buds or cuttings, which usually come true to type, although in some cases a certain percentage may revert to the normal green form. The majority of variegated forms do not come true from seed, but exceptions may be noted, as in the variegated nasturtium, in *Ptelea trifoliata aurea,* etc. Color types in corn and some other species are inheritable and, according to Lindstrom (1918), are transmitted in strictly Mendelian ratio. He recognized the following seedling types: white or albino, virescent white and yellow; and golden, fine green-striped, Japonica white-striped and Japonica yellow-striped as mature plant types. Variegated forms generally appear quite healthy, but it has been noted that they are not so hardy as their normal green progenitors. They are reported to succumb more readily to unfavorable conditions, such as drought or cold (Clinton, 1914) and in some cases to be more susceptible to the inroads of parasitic forms.

Types of Virus Diseases.—No satisfactory classification of the virus diseases has yet been presented. It has been customary to apply names on the basis of hosts attacked or of symptoms, but such a classification is inadequate. If a virus is a distinct entity, as distinct as and no more variable than a bacterial or fungous entity, a logical classification may be forthcoming (Johnson, 1927; Quanjer, 1931). On the basis of present information, certain groups may be recognized which represent single virus entities, related or similar viruses or groups of viruses. With present information, it is not possible to make a certain assignment of some of the recognized diseases. Some of the more important groups are as follows:

1. Infectious chlorosis, general chlorosis or variegation transmitted only by organic union as in budding or grafting but not contagious.

2. The peach group including yellows, little peach, peach rosette and phony peach disease, apparently four different entities transmitted by organic union but spreading in nature by unknown agencies.

3. Wheat mosaic and rosette transmissible to all species of the tribe Hordeæ and in nature communicated in some way through the soil.

4. Curly top of beets and many other cultivated crops as well as numerous wild plants. In nature, transmitted by the leaf hoppers *Eutettix tenellus* and *Agallia sticticollis.*

5. Aster yellows transmitted by the leaf hopper *Cicadula sexnotata* to numerous hosts, including aster, celery, lettuce, etc.

6. Potato mosaics, including several distinct viruses or virus complexes, transmitted mainly by aphids.

7. Potato leaf roll, a single virus entity transmissible to some other Solanaceæ.

8. Tobacco mosaics, including a group of distinct or closely related viruses.

9. Tobacco ring spot transmissible to other Solanaceæ and many other unrelated species in 16 different families.

10. Bramble viroses, caused by a group of virus entities, affecting raspberry, blackberry and loganberry.

11. Strawberry viroses, caused by several apparently distinct virus entities.

12. Cucumber or cucurbit mosaics, including a group of distinct viruses affecting various cucurbits and also some species from other families.

13. Legume mosaics, including a number of distinct or closely related viruses, transmissible by the seed.

14. Hop viroses, including several viruses, apparently confined to the nettle family.

15. Bulb mosaics or other viroses, including a number of viruses not yet well differentiated.

16. Bunchy top, affecting banana, plantain and Manila hemp, probably caused by a single virus.

17. Grass mosaics, affecting various wild or cultivated grasses, sugar cane and corn, probably includes several distinct viruses.

18. Grass streaks, affecting corn, sugar cane and various other Gramineæ, probably including several either distinct kinds or strains.

In addition to the viruses included in the groups enumerated, the following appear to represent distinct viruses: cotton leaf curl, reversion or nettlehead of currants, cranberry false blossom, potato spindle tuber, spotted wilt of tomato, curl disease of beet, spike disease of sandal and pineapple yellow spot. There are other less known virus diseases in which the virus may prove to be either distinct or referable to some of the recognized entities.

Certain virus diseases have been shown to represent double infections, *i.e.*, the combined action of at least two distinct viruses—*e.g.*, the streak of tomato due to the latent virus of healthy potato combined with tobacco mosaic. Potato latent in combination with tobacco vein-banding virus appears to give rugose mosaic symptoms on potato, and other mixtures are known. It seems probable that other viruses now considered to be caused by single virus entities will be resolved into two or even more separate entities as methods of separation and purification become perfected.

The Infectious Nature of Virus Diseases.—The mosaic disease of tobacco was the first virus disease that was proved to be transmissible from diseased to healthy plants. The discovery was made by Iwanowski (1892) that extracted juice of a tobacco plant affected with mosaic would infect a healthy plant if pricked into its tissues, even though it had been passed through a Chamberland filter. Similar results were later obtained by Beijerinck (1898), and the filterable character of the infective principle of tobacco mosaic and other viroses has since been repeatedly demonstrated. The first record of insect transmission of a virus disease is in the work of Takami (1901), who connected the "stunt" disease of rice with the leaf hopper *Nephotettix apicalis.* The beet leaf hopper (*Eutettix tenellus*) was proved by Ball (1906–1909) and Shaw (1910) to be the vector for beet curly top (see this disease). The transmission of tobacco mosaic by aphids was first demonstrated by Allard (1914). The connection of aphids with potato leaf roll was pointed out by Botjes (1920) and confirmed a few years later by Schultz and Folsom (1923, 1925) and Murphy (1923). Since that time, rapid progress has been made in the determination of the insect vectors of the various viroses.

Methods of Transmission.—Virus diseases show different degrees of infectiousness and consequently may be transmitted in different ways in nature or artificially:

1. Transmitted by budding or grafting only: infectious chloroses, and the peach group of viroses, potato witches' broom, hop mosaic (?), spike disease of sandal, etc. The peach viroses spread in the orchard, but the method of contagion is unknown.

2. By a specific insect, by several insects or also by budding or grafting: aster yellows, cranberry false blossom, curly top of beet, potato leaf roll. (A very low percentage of juice inoculations of curly top were successful.)

3. Transmitted by the juice of a diseased plant, by grafting or budding and also by insect vectors. This includes most mosaics, and the most important vectors are species of aphids.

4. Transmitted by the seed. Most of the virus diseases are not perpetuated by the seed (true seed) from infected parents; nevertheless, seed inheritance has been found to be general for legume mosaics and occurs also in lettuce mosaic and petunia mosaic. No satisfactory explanation has been offered for the general inheritance of mosaic among legumes and its absence in most other families.

Tobacco mosaic is the most infectious of all the virus diseases, and its extremely infectious nature may be illustrated by the fact that it can be readily communicated by touching a diseased plant and then touching a healthy plant. The virus or infective agent is able to retain its infective properties when dried. Diseased leaves when dried and ground to a powder will still communicate the disease after months of desiccation.

The virus may be extracted from diseased tobacco leaves by ether, chloroform, carbon tetrachloride, toluene or acetone without the destruction of its infective properties. An infinitely small amount of the virus when introduced into a susceptible plant will increase rapidly until it has spread throughout the entire structure and consequently must have increased many fold.

Insect Vectors of Virus Diseases.—The insects responsible for the transmission of virus diseases may be divided into two groups: (1) biting and (2) sucking. The first are relatively unimportant, only a few authentic cases being recorded: cowpea mosaic by the bean leaf beetle, cucumber mosaic by the two cucumber beetles and potato spindle tuber by grasshoppers and a few beetles. In the case of biting insects, there seems to be no specificity in the transmission, the transfer or inoculation being mechanical. The principal vectors belonging to the second group are (1) thrips, as *Frankliniella insularis* (spotted wilt of tomato), and *Thrips tabaci*, connected with the transmission of several different diseases; (2) leaf hoppers, as *Eutettix tenellus* (curly top), *Cicadula sexnotata* (aster yellows) and several others; and (3) plant lice or aphids known to be responsible for the transmission of numerous diseases. At least 23 species are known to be vectors of 27 or more viroses on numerous plants. The aphids are clearly the most common and efficient vectors of virus diseases. Besides insects of the three groups mentioned, vectors have been reported from the lace bugs, capsid bugs, mealy bugs and white flies (see Smith, 1931).

Some of the more important factors in the relationship between viruses and insect vectors may be briefly enumerated: (1) Numerous cases are on record of a delay in the development of the infective power (a period of incubation) following feeding upon a diseased plant; (2) there is an apparent specificity of certain insects for particular viruses, as illustrated by diseases in which only a single species of vector is known or by those in which certain vectors are much more efficient than others; (3) no morphological or cytological differences between viruliferous and non-infective insects have been discovered; (4) many vectors retain their infective power for a long time or even during their whole lifetime, indicating the possibility of an increase of the virus within the body of the insect; and (5) the virus is not transmitted by young from the eggs of viruliferous parents until after they have fed upon infected plants. The evidence appears to indicate that the relationship between vector and virus is something besides a mere mechanical connection.

It is also of interest to note some of the relationships between the plant and the insect vectors: (1) There may be a selective transmission; *i.e.*, a certain insect may transmit only one virus after feeding on a plant containing a mixture; (2) the virus exists in all parts of a plant, but it cannot be obtained by a vector from all parts with equal ease; (3) a virus

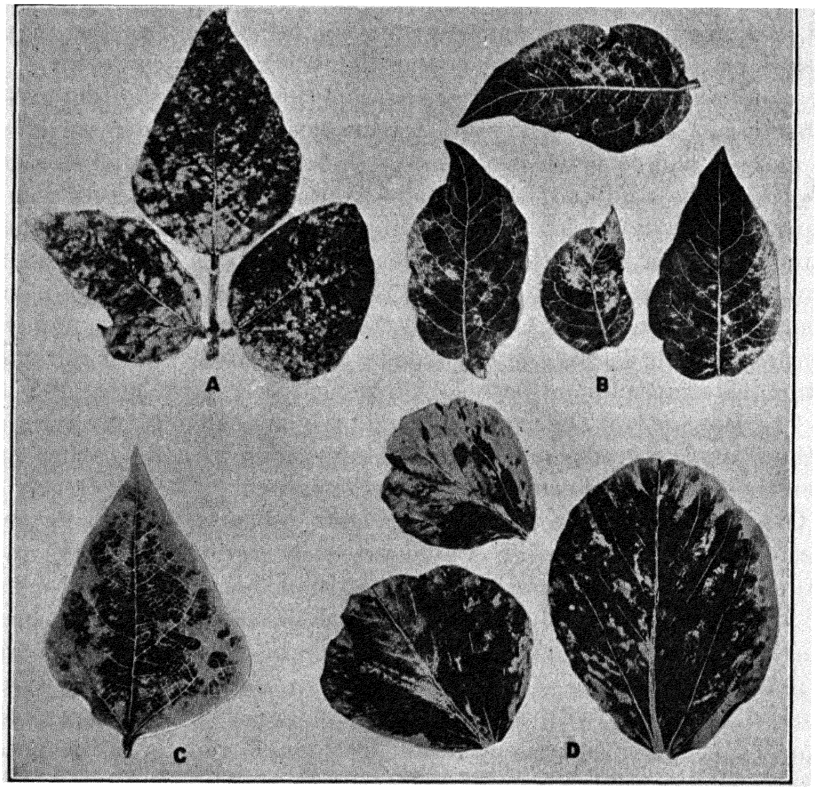
may exist in a plant without causing any external evidence of its presence, and such symptomless "carriers" may yield the virus to insects feeding upon them; and (4) infection may result from the feeding of a single viruliferous insect, but infection is greater with large numbers of vectors.

Mosaic Diseases.—The name "mosaic disease" is a direct translation of the German name "Mosaikkrankheit," which was first given by Mayer in 1886 to a disease of tobacco which has since been known by that name. All mosaic diseases possess two distinctive characters: (1) the mottling of the plant, due to alternating patches or spots of light green or yellow and dark green, although under certain conditions the mottling may be masked or not evident; and (2) the highly infectious character of the juices of diseased plants even after filtration through mantles that exclude all bacteria. During recent years, the increasing economic importance of the mosaics and other virus diseases of our crop plants has given a great impetus to the study of these obscure troubles, with the result that the known mosaics have been increased from the single one affecting tobacco to numerous mosaics of other Solanaceæ, Cucurbitaceæ, Leguminoseæ and Gramineæ with scattered cases in 20 or more additional families of both Dicots and Monocots. Current literature is replete with numerous reports of mosaic diseases upon some new plants, either wild or cultivated. Future historians of plant pathology may characterize the present time as the "mosaic age," or the period marked by increased and intensive study of the virus types of plant disease, as contrasted with the earlier times when the study of fungous diseases predominated.

General Appearance of Mosaic Plants.—Plants which contract a mosaic disease early in life are reported by various observers to be distinctly *paler in general appearance* than normal healthy plants, due to a lessened production of chlorophyll, while late, or primary, infections may affect only the youngest parts or may not be evident until the next season. *General reduction in size* is frequently a pronounced symptom, with spindliness as an occasional accompaniment. Dwarfing is shown in extreme form in "mosaic dwarf" of the potato, an advanced or severe form of potato mosaic. Dwarfing is also characteristic of the mosaic of raspberries, peas, kidney beans, clovers except sweet clover and certain varieties of Canada field peas. *Excessive branching* sometimes accompanies the dwarfing. *Premature yellowing and dropping of the leaves*, especially the lowermost, occur in the mosaic of beans, tomatoes and some phases of potato mosaic, while localized *necrosis* of tissue may occur in either leaves or stems, although this is not of common occurrence. In the Pacific Northwest, under upland conditions with extreme drought, premature death and browning of the chlorotic areas of the leaves have been noted in potato mosaic, followed by early death of the entire plant. In the streaking phases of rugose mosaic of the potato, necrotic areas develop in leaves, petioles and stems,

while dropping of the lower leaves is a characteristic of "leaf drop" (Murphy) and "russet dwarf" (Hungerford) of potatoes.

Effects of Mosaic on Leaves.—Leaf changes which point to the presence of mosaic are (1) mottling or the occurrence of a grouping of light-green or yellowish- and dark-green areas, the spots varying in form and size from irregular to angular and from small to large, certain patterns being characteristic of specific mosaics; (2) curling and ruffling, due to unequal development of larger leaf areas; (3) savoying



of pea bean showing blistered type; *D*, mosaic of Windsor bean. (*After B. T. Dickson, Macdonald Coll. Tech. Bul.* **2.**)

or blister-like elevations with intervening depressions, the latter being occupied by the chlorotic areas when mottling is evident, thus giving the leaf surface a puckered, wrinkled or rugose surface; (4) dwarfing and distortion, sometimes the former alone but frequently accompanied by malformations, such as asymmetric development of the laminæ, dissection of the margin or reduction of the leaves to little more than midribs. The extreme types are illustrated by "filiform leaf" and "fern leaf" of tomatoes, "filiform leaf" of sweet potato and the long, sinuous or ribbon-like leaves of tobacco plants. Extreme discolorations

are most likely in the case of early or seedling infections and may be aggravated by unfavorable environmental conditions.

Effects of Mosaic on Flowers.—Several different effects may be noted: (1) dwarfing of entire flower or of parts; (2) distortion or malformation of parts; (3) mottling of the corolla; and (4) the fall of blossoms. The effects may be illustrated by the dwarfed and paler blossoms induced by cucumber mosaic; dwarfed corollas sometimes occurring in tobacco mosaic; change of a gamopetalous corolla to a pseudopolypetalous form, an observed effect in some cases of tobacco mosaic; mottling in the flowers of sweet peas, petunias and tobacco; and dropping of the blossoms in tomatoes, beans and potatoes.

Effects of Mosaic on Fruits.—The most important fruit effects are (1) mottling, russeting or spotting; (2) dwarfing and distortion; and (3) a reduction in the quantity of seed formed and also in its vitality. Mottling has been noted in the fruits of tomatoes, peppers, cucurbits and beans; russeting and spotting in tomato fruits; dwarfing and distortion in tomatoes, beans, soy beans and cucumbers; and reduction of yield of seed and loss of viability in corn, soy beans, peas and clover. The effects of mosaic on the fruits of the cucumber are very pronounced and characteristic. In addition to the mottling,

> . . . they show swellings of all sizes, some isolated and others merging into one another in such a way as to produce very irregular forms, the symptoms often being well defined on fruits which are but an inch in length. The fruits of this type have given rise to the names "wart disease" and "nubbin," which have been applied to the disease by growers in some parts of the country.
>
> In later stages of the disease, the vines occasionally produce fruits which are smooth, pale, whitish green in color and rather more blunt at the ends than normal fruits of the same age. In most cases, these fruits are mottled with fine spots of yellowish green, and a few dark-green projections appear here and there on the surface. These are usually small, but occasionally fruits are found which have a single large, dark-green swelling near the stem end, producing a most unusual appearance.
>
> These white fruits are responsible for the older common name of the disease, "white pickle," which was the term applied to the trouble by the growers in Michigan and Wisconsin for some years (Doolittle, 1920).

Effects of Mosaic on Stems.—The observed effects of mosaic on the stems may be grouped as (1) discoloration, including general pallor, mottling and striping or streaking; (2) dwarfing, involving a reduction in diameter and in length of internodes; and (3) the formation of necrotic lesions or cankers. Although herbaceous stems are chlorophyll-bearing, mottling in them is rather rare but has been noted in tobacco, tomato and squash. Dwarfing varies all the way from practically normal stems to others very much reduced, the extreme resulting generally from early severe infections. Lesions in the stems of mosaic-sick corn and

sugar cane have been shown to occur as secondary stages of mosaic, while the streaking of potato stems affected with some types of rugose mosaic has been noted.

Pathological Histology of Mosaic Plants.—Supplementing earlier studies with tobacco, tomato and potato, Dickson (1922) has studied the microscopic characters in a large number of plants affected with mosaic and has shown that certain features are common to all.

It is not to be understood, however, that in any leaf section from a diseased plant all characteristics equally well developed are to be observed. Much depends upon the growth rate and environment of the host plant.

The following are the most important of the features which have been noted: (1) A reduction in the leaf thickness of the chlorotic areas due to hypoplasia of both spongy and palisade tissue, but especially the latter. The general ratio of thickness between the light-green and dark-green areas of leaves is 2:3. In the extreme cases, the palisade layer may be reduced to a single row of nearly cubical cells, but there are all gradations from this condition to nearly normal. (2) A more uniform and compact arrangement of the mesophyll cells in the light-green areas, with less intercellular space. This makes the light-green areas more transparent than the darker areas. (3) In the light-green areas the chloroplasts may be paler than normal but in usual numbers, while in more severe infections they may be reduced in number and in size with much diminution in color. In very acute infections the chloroplasts may coalesce into irregular green masses, or they may become completely disintegrated into small, hyaline bodies. This disintegration of the cell contents may produce the dead flecks or spots which are especially noticeable under conditions of prolonged or severe drought. (4) The cells of the dark-green areas are larger than normal, contain more chloroplasts and the chlorophyll is also darker than in normal or healthy leaves.

When savoying is very marked the palisade cells are in a condition of hyperplasia. Either they have divided to form two layers of palisade cells or they are narrower, longer and more numerous or both may have occurred (Dixon, 1922).

(5) The volume of intercellular space in the dark-green areas is increased, and this feature, together with the greater depth of color of chlorophyll, makes these areas look darker than normal (see Fig. 85).

It has recently been pointed out (Rochlin, 1930) that phloëm necrosis which has been recognized as an important character of leaf roll of potato also occurs in potato mosaics. Three types of phloëm alterations are recognized: (1) *necrobiosis*, characterized by gradual swelling of the cell walls, etc., and occurring in either healthy or diseased plants; (2) *phloëm necrosis*, correlated with leaf roll and other virus diseases; and (3) *obliteration* of phloëm, typical of senescence.

In virus diseases in general, the most prominent effects are (1) chlorosis, localized or general; (2) dwarfing of the entire plant or hypoplasia of certain tissues; (3) localized or general collapse and death of tissues or organs, with spot necrosis, phloëm necrosis or streak, in the extreme cases resulting in death of the plant; and (4) more rarely in localized hypertrophy with gall formation, as in Fiji disease of sugar cane or general overgrowth, as in giant hill of the potato.

The Nature of the Causal Agency in Virus Diseases.—The numerous studies which have been carried out since the recognition of mosaics and related diseases have failed to give positive evidence as to the real nature of the causal agency. An infectious disease, without any visible causal organism, and transmitted by an agent or principle so small that it will pass through ordinary bacteria-proof filters, presents a field of investigation that is taxing the ingenuity and imagination of scientists. Speculation and theorizing have marked the progress of our knowledge of the virus diseases; but as time has passed, more careful attention to corroborating facts has been given. Of the various possible views as to the causal agency, many have been exploited and discarded, but the four following theories are given attention in current literature: (1) the bacterial theory; (2) the enzymatic theory; (3) the virus theory; and (4) the protozoan theory.

The Bacterial Theory.—The fact that the various virus diseases behave much like known bacterial diseases gave the earlier workers a prejudiced outlook, and they naturally expected to find bacteria in plants affected with mosaic. The bacterial theory was supported by Mayer (1886) in his study of tobacco mosaic. Ivanowski (1903) and Hunger (1905) found bodies which they believed to be bacteria and also amœba-like structures in diseased cells. Boncquet (1916) found bacteria, a small nitrate-reducing streptococcus, in mosaic-affected tobacco and later (1917) gave the name of *Bacillus morulans* to a form which he believed to be the cause of the curly top of sugar beet. Dickson (1922) still inclined to the bacterial theory and reported the finding of bacteria-like bodies, as had Iwanowski. Without entering into details, it may be stated that none of the investigations recorded has presented tests or experiments which have given convincing evidence of the causal relation of bacteria to any virus trouble. The careful technique of recent investigators has practically excluded bacteria as possible causal agents. It may be stated, however, that the recent work of Hadley, Kendall and others on bacterial life cycles and the filterability of certain bacteria points to the possibility that plant viruses may yet be found to have a connection with known forms of organisms.

The Enzyme Theory.—The enzymatic nature of the mosaic disease of tobacco was first proposed by Woods (1899 and later). He suggested that the chlorotic condition of mosaic leaves was caused by an abnormal

and extensive development of oxidizing enzymes. His view was adopted with some modifications by later workers, and even as late as 1917 Freiberg accepted and supported the enzyme theory, although he did not consider the active enzymes to be of the nature of oxidases. That the infective agency could not be an oxidase was convincingly demonstrated by Allard (1916), who showed that the oxidase could be destroyed by hydrogen peroxide without destroying the infective properties of the juice and that the active agency of tobacco juice may be destroyed without the destruction of the oxidase action. The enzyme viewpoint was supported in part by (1) the absorption of the active principle by talc, a characteristic of colloidal compounds, including enzymes; (2) a specificity of reaction between the infective principle and formaldehyde; (3) the resistance of the agent to numerous antiseptic substances; (4) the destruction of the infectious properties by concentrations of alcohol that inactivate enzymes; and (5) the destruction of the infective properties by the same temperatures that inactivate enzymes. While an enzyme might explain the hypoplastic chlorotic areas, it is difficult to understand how such a compound could be responsible for the hyperplasia of the dark-green areas or for hyperplastic developments in virus diseases other than the mosaics. The enzyme theory also involved the assumption that a small quantity of enzyme introduced into normal cells must have the power to start the production of this same enzyme and that this operation once started could not be stopped. At the present time, most of those who have been making a special study of mosaic or other virus diseases are unwilling to accept the enzyme theory.

The Filterable-virus Theory.—The term "virus" as now used in relation to plant diseases is understood to mean the filterable agent or principle which has the power of inducing the disease. The use of the term does not carry with it any preconceived concept as to the nature of this principle, and different views have been held by various investigators.

The idea of the virus as a non-corpuscular, water-soluble substance was apparently the concept of Beijerinck (1899) in the use of the term *contagium vivum fluidum*. The second idea of the virus was that of a minute, bacteria-like organism, so small as to be beyond the range of miscroscopic vision, a so-called "invisible microorganism." At first, the ability of the agency or virus to pass through the pores of a standard Berkefeld or Chamberland filter was not opposed to the concept of the virus as a bacteria-like organism, but later tests showing the extreme minuteness of the infective particles as shown for tobacco mosaic (Duggar and Karrer, 1921, 1923) have taxed the imagination to form a new concept for the causal agent if it is really an organism. Previous to the ultra-filtration experiments of Duggar and Karrer, the capacity of certain filters (the Livingstone atmometer cup or a layer of powdered talc 7/8 inch thick) to prevent the passage of the virus of tobacco mosaic had been

demonstrated by Allard (1915), and Doolittle (1920) had shown that the cucumber mosaic virus passed through a Berkefeld but not through a Chamberland filter. The opinion of Allard as to the nature of the infective agent, based on his studies of tobacco mosaic (1916), was that

> A specific, particular substance not a normal constituent of healthy plants is the cause of the disease. Since this pathogenic agent is highly infectious and is capable of increasing indefinitely within susceptible plants, there is every reason to believe that it is an ultramicroscopic parasite of some kind.

By the use of a series of special filters, Duggar and Karrer (1921) have arrived at a conclusion as to the size of the mosaic particles in tobacco mosaic, a comparison being made with the particles of hemoglobin, the approximate size of which was known:

> Assuming that at most the hemoglobin particles worked with may have possessed a diameter of 30 millimicrons, more or less, and that the average small diameter of bacterial plant pathogenes is around 1000 millimicrons (1 micron) (some being as low as 500 and others as large as 1500 millimicrons), we have 30:1000 to express roughly the diameter relations of mosaic-disease particles in comparison with bacterial plant pathogenes. On the basis of this average relation it is interesting to note that the volume relation would be about as follows: 1:37,000 or about 26:1,000,000, assuming that in each case we may treat the bodies as spherical structures.

As a result of further studies concerning the properties of the mosaic virus, the details of which must be omitted, Duggar and Karrer (1921) have advanced a new concept of the nature of the mosaic particles. They suggest that

> The causal agency in mosaic disease may be, in any particular case, a sometime product of the host cell; not a simple product such as an enzyme, but a particle of chromatin or some structure with a definite heredity, a gene perhaps, that has, so to speak, revolted from the shackles of coordination and, being endowed with a capacity to reproduce itself, continues to produce disturbance and "stimulation" in its path, but its path is only the living cell.

May it be that particles of the living substance, when controlled and dominated by the parent living substance, are held in the shackles of coordination but when set free from this domination and subjected to the forces of foreign protoplasm continue in uncontrolled riot?

One of the objections to the microbic character of the mosaic virus has been the failure to increase this in artificial cultures. Recent cultures have been reported by Olitsky (1924), from which he concludes that "the incitant of mosaic disease of tobacco and tomatoes is a living microbic body which can be cultivated in an artificial medium." The evidence, however, does not seem entirely convincing, and the multiplication of viruses outside living organisms has not been substantiated by other

investigators. The phenomena of symptomless carriers and attenuation of plant viruses are suggestive when contrasted with a similar behavior of bacterial pathogenes.

The Protozoan Theory.—Amœboid bodies were first described by Iwanowski (1903) as occurring in the cells of mosaic tobacco plants; and the same year, small bodies, now called "Negri bodies," were discovered by Negri in the brain cells of animals suffering from rabies (McKinley, 1929; Kunkel, 1928). The character of these bodies suggested that they might represent stages in the life cycle of a protozoan parasite, and the idea gave impetus to the study of numerous other animal and plant virus diseases by careful cytological methods, with the result that, wherever careful studies have been made, similar plasma-like bodies have been found in the tissues of many other animals and plants affected with virus diseases. Such findings have been reported by Lyon (1910), McWhorter (1922) and Kunkel (1924) for the Fiji disease of sugar cane, by Matz (1919) for the mosaic of sugar cane, by Kunkel for the mosaic of corn (1921) and Hippeastrum (1922), by McKinney *et al.* for wheat rosette and mosaic (1923), by Goldstein (1924 and 1926) and Rawlins and Johnson (1925) for tobacco mosaic, by Smith for potato mosaic (1924), by Eckerson for tomato mosaic (1926) and by Goldstein for dahlia mosaic and dwarf (1929), and their presence has more recently been noted in other virus diseases. These bodies were first called x-bodies by Goldstein in 1924 and since that have generally been referred to under that name. The amœboid changes of these bodies, their division and migration have been described in a number of cases, but while suggestive of protozoan character, their real nature and significance are still uncertain. The general opinion is that the x-bodies do not represent stages in the life cycle of a causal organism but are rather the results of derangements in cells induced by the operation of viruses.

The work of Nelson (1922) is a conspicuous landmark in the march of progress in the study of virus diseases. When first presented "the occurrence of Protozoa in plants affected with mosaic and related diseases" made quite a sensation. The known occurrence of flagellates in the latex vessels of certain spurges (Euphorbiaceæ) and milkweeds (Asclepiadaceæ) was probably the guiding star in Nelson's work. He described and presented illustrations of what he believed to be a new form of biflagellate from the phloëm of bean and clover plants affected with mosaic and structures which he identified as typical trypanosomes from the sieve tubes of tomato plants suffering from mosaic and also from potato leaf-roll plants. The fame of the discoverer was short-lived, for it was soon found that the structures described as Protozoa were normal inclusions in healthy as well as diseased cells (Duggar and Armstrong, 1923; also other papers published in *Phytopathology* **13**: 324–333 (1923) and had been described years before by Strasburger.

It should be of interest to note that a number of workers have been sufficiently convinced of the protozoan or organism nature of the intracellular bodies accompanying virus diseases to apply definite binomials to them. Mention may be made of *Northiella sacchari* by Lyon for Fiji disease of sugar cane (1921), *Strongyloplasma iwanowskii* by Palm for tobacco mosaic (1922) and *Plasmodiophora solani* by von Brehmer and Bärner (1930). It is difficult to understand why Nelson (1922) and Eckerson (1926) did not also build binomial monuments to mark their discoveries.

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INFECTIOUS CHLOROSES

The chloroses of plants not the result of environmental factors may be (1) variegations or non-infectious chloroses perpetuated either by seed or by vegetative propagation; (2) chloroses transmitted only by

graft union of chlorotic and green parts, the "infectious chloroses"; and (3) infectious chlorotic conditions transmitted by juice or insects referable to the mosaics.

History.—The transmission of variegation by grafting has been known since the successful experiments of Wats in 1700. First real importance of this peculiarity followed the introduction from the East Indies in 1868 of *Abutilon striatum*, a form with beautiful variegation. This attractive plant was widely propagated in France and England, and in 1869 Lemoine transferred this leaf variegation to other species of Abutilon by grafting, but the true nature of the peculiarity was not suspected until 1899 when Beijerinck suggested that it was like the mosaic of tobacco. Variegations other than those of Malvaceæ were shown by Masters (1869) to be communicable. The most extensive work on transmissible variegation was begun in 1872 by Lindmuth and continued interruptedly until 1907. Important contributions were made by Baur (1904, 1908) when the relationship of infectious chloroses to the virus diseases was realized. Later contributions of special merit have been made by Hertzsch (1927), Rischkow (1927) and Davis (1929).

Symptoms.—The amount of variegation and the exact patterns vary in the different species or even in the same species under different influences. Küster (1927) states that infectious chloroses show generally irregular limitation of the chlorotic areas as contrasted with sharply limited areas in the non-infectious types. The variegation may vary from forms like *Abutilon indicum*, showing "a single more or less expanded yellow spot in the leaf with little of the green remaining to others with only a slight yellowing along veins or at the tips of marginal leaf teeth as in *Sorbus aucuparia*, or the leaves may show large or small yellow spots with a typical mosaic-like character in certain cases." In some cases, affected leaves may remain smaller than normal ones and show some wrinkling or rugosity. In cases showing the most extreme reduction of the chlorophyll-bearing surface, the plants may be killed because of inhibited photosynthesis.

Etiology.—The chlorosis or variegation of the foliage in this group may be transmitted from chlorotic stock to normal green stock by budding or grafting or even in some cases by transplanting a piece of diseased cortex in the cortex of a normal plant. Morren (1869) reported transmission of infection in some cases by inserting a petiole with a variegated leaf attached into an incision in the bark of a normal green plant; but, in general, actual organic union between chlorotic and normal tissues appears to be necessary for the transmission of the chlorotic disturbances. The establishment of the graft upon the scion appears to be the important feature, but variegation is not transmitted in all such cases, some types showing, however, a much higher percentage of successful transfer than others. All kinds of inoculations using juice, filtered or unfiltered, or macerated or ground tissue have failed, even the recent very painstaking trials of Davis (1929). It has also been found impossible to obtain transfer with insects (Rischkow, 1927).

The following data are based largely on the contributions by Baur (1906–1908):

There are no external characters which indicate whether a given form is infectious or will behave like ordinary variegation. For example, of three variegated varieties of privet (*Ligustrum vulgare*)—(1) *albomarginatis*, with white leaf margins; (2) *aureum*, a typical golden form; and (3) *aureovariegatis*, with yellow spotted leaves—only the last proved to be infectious, while the two others were of the non-infectious type.

Buds of *Cytisus hirsutus* transplanted to *Laburnum vulgare chrysophyllum* became chlorotic the same as the stock. A peculiar response was noted when the variegated burning bush was grafted on the normal green variety or the normal on the variegated variety. A chlorotic condition was transmitted in each case, but the pattern was different, showing, instead of golden margins, a yellow veining of the leaves. This new pattern remained constant when propagated by cuttings.

It has been shown that the virus or the infective principle is produced in the variegated leaves under the action of direct sunlight. If the old variegated leaves of an Abutilon or any other species showing infectious chlorosis are darkened, the new leaves that appear at the growing point will be pure green even though exposed to the light, but darkening of the growing point alone does not prevent the variegation in the newly formed leaves. Partial darkness seemed to have the same effect upon the production of the virus as complete darkness.

A peculiarity of the virus or infective principle is cited by Baur (1906). *Abutilon thompsonii*, a variegated form, and a normal green *A. indicum* were grafted on adjacent branches of *A. arboreum*, a species immune to infectious chlorosis. The new shoots of *A. indicum* became chlorotic, indicating that the infective principle was transmitted through the tissues of the immune stock. In similar tests with an immune Lavatera, the virus was not transmitted. In no cases of infectious chlorosis is the virus transmitted to the embryo plant in the seed, as numerous tests have shown that seeds from chlorotic parents yield normal green seedlings.

It has been pointed out (Rischkow, 1927) that both infectious and non-infectious chloroses may be associated in the same plant. In *Euonymus japonica*, for example, infectious chlorosis may be associated with other non-infectious variegations of the "marmor," "chlorinomarginata" and "aureomaculata" types, thus masking or obscuring the infectious type. Separation of the two can be accomplished by grafting the variegated form on one having normal, uniformly green leaves.

Observations and transmission experiments have demonstrated two distinct types of infectious chlorosis in the mallows: (*a*) in *Abutilon striatum thompsonii*, showing as a yellow spotting of the leaves and a yellow coloration of the veins; (*b*) in *A. darwinii tesselatum*, characterized by pale green spots and stripes on the leaves, but with the veins green.

Lavatera arborea is immune to the *a* type but very susceptible to the *b* form, which causes complete deforming of the leaves, followed by death, while other Malvaceae are susceptible to both types (Hertzsch, 1927).

Hosts.—According to Davis (1929), "infectious chlorosis is known to occur among eight families, eighteen genera and roughly among thirty-five species." The following are some of the more important known cases of infectious chlorosis:

1. Mallow family (Malvaceæ): Abutilon (*Abutilon striatum* var. *thompsonii*). The variegation of this variety is transmissible to other species and varieties of Abutilon and also to species of several other genera of the family.
2. Staff-tree family (Celastraceæ): Japanese burning bush (*Euonymus japonica*), represented by the horticultural varieties *argenteo marginatus*, *aureomarginata*, *marmor*, *chlorinomarginata* and *aureomaculata; E. radicans.*
3. Olive family (Oleaceæ): *Fraxinus pubescens aucubifolia*, a cultivated ornamental with bright yellow, blotched leaves; variegated privets (*Ligustrum vulgare aureum*); *L. vulgare aureo-variegatum* and *L. vulgare albo-marginatum;* jasmine, *Jasminum officinale variegata* and *J. revolutum aureovariegata.*
4. Pea family (Leguminosæ): Golden chain (*Laburnum vulgare chrysophyllum* and *L. vulgare aureus*).
5. Dogwood family (Cornaceæ): Tartarian dogwood (*Cornus alba* and *C. alba argenteo variegatum elegans*).
6. Rue family (Rutaceæ): Hop tree (*Ptelea trifoliata aurea* and *P. trifoliata variegatis*).
7. Rose family (Rosaceæ): European mountain ash (*Sorbus aucuparia dirkenii aurea* and *S. aucuparia leuteo-variegatum*).

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PEACH YELLOWS

This disease of the peach and some related trees is characterized by pronounced yellowing of the foliage, production of wiry, sickly shoots, premature ripening of the fruit and invariably terminates in the death of the affected tree.

History and Geographic Distribution.—Peach yellows is confined entirely to America. It was first noted in the vicinity of Philadelphia and was the subject of a special paper by Judge Peters in 1806. There are authentic records of its occurrence in 1791 and less reliable evidence of its existence as early as 1750–1760. In 1806–1807, the disease was apparently confined to the Philadelphia section, which included adjacent territory extending into New Jersey and Delaware. From this time on, the disease spread northward and northeastward, reaching Connecticut by 1814 and Massachusetts a few years later; westward and northwestward, being first reported from western New York about 1824, from Ohio in 1849, from Indiana about the same time, from Michigan in 1866–1867 and from Ontario in 1878; southwestward through Delaware and Maryland, its progress being somewhat slower, but it finally reached Arkansas and northeastern Texas. The present range of yellows includes the territory from Massachusetts south to the Carolinas, the southern boundary including most of Tennessee and crossing Arkansas and Oklahoma, the northern boundary crossing New York, the peach districts of Ontario and Michigan and the western extent reaching into Missouri and Kansas. True yellows has not been reported from regions west of the Rocky Mountains, California yellows or little leaf being a disease of a different nature.

The contagious character of yellows was recognized in 1806 by Judge Peters, who said: "I find that sickly trees often infect those in vigor near them by some morbid effluvia." The first thorough study of the disease was published by Smith (1888), and this exhaustive preliminary report was followed by several others by the same writer (1889, 1891, 1893, 1894). Yellows was also studied by workers in several of the states: Bailey in New York (1894), Selby in Ohio (1896, 1898), Clinton in Connecticut (1909), Blake in New Jersey (1910), Caesar in Ontario (1912) and Atwood in New York (1914). More recently, special investigations have been made on the disease in New Jersey (Cook, 1921; Blake, Cook and Conners, 1921), while control has been emphasized by McCubbin in Pennsylvania (1924, 1928).

Peach yellows is recognized as an exceedingly serious disease in much of the territory recorded, being especially destructive in Pennsylvania, New Jersey and the peach belt along the shore of Lake Michigan.

"There has been a noted rise and fall in the intensity of yellows, the disease assuming the nature of an epidemic every 10 or 15 years, with quiescent periods intervening between the periods of excessive virulence" (McCubbin, 1924). In the eastern United States, serious outbreaks have occurred in 1791, 1806–1807, 1817–1821, 1845–1858, 1874–1878 and 1886–1888.

Symptoms and Effects.—Certain symptoms may be regarded as unquestionably indicating the presence of yellows, while others point to the existence of either yellows or little peach, a disease of somewhat similar nature.

1. *Fruit Characters.*—In a bearing orchard, the most reliable diagnostic character is the *premature ripening* of the fruit, which may vary from a few days to 3 weeks. The prematurely ripened fruits are frequently, though not always, *abnormally large*, are more watery in texture, have an *insipid flavor* and are often *speckled or blotched with red.* The coloration is quite different from that of normal mature fruit and may be largely confined to the skin, or internal spots and streaks may be prominent, while the flesh may be much more prominently marked with red around the pit than in normal fruit. The amount of red coloration is variable, ranging from slight to spotted, mottled or almost completely crimson. Fruit pre-

maturely ripened as a result of borers is never red spotted, and the characteristic wiry shoots are lacking. It should be noted that the yellows causes but little abnormal coloration of the Elberta. Abnormal fruits may appear on a single limb or only on a part of a limb, or in many extreme cases all of the fruits may be prematurely ripened.

2. *"Willow" or "Broom" Shoots.*—The production of slender, much-branched, more or less erect, wiry shoots clothed with small yellow leaves often spotted with red is a symptom of equal value to the abnormal fruit,

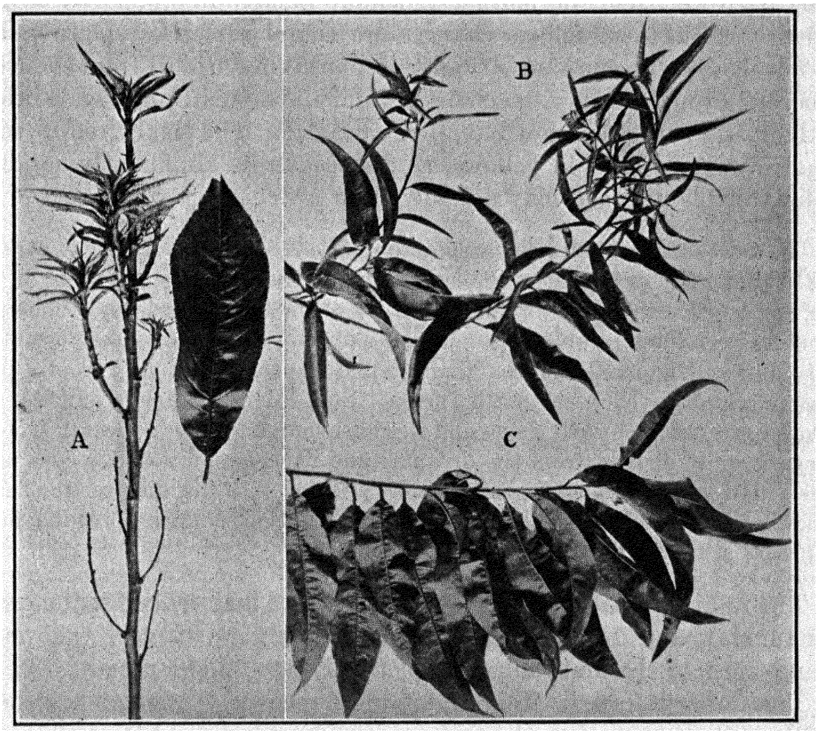

FIG. 74.—Peach yellows. *A*, typical "willow" shoot showing the upright, much branched habit with small, yellow foliage, with normal leaf for comparison; *B*, terminals showing slender growth, narrow leaves, upright habit; *C*, healthy shoot for comparison. (*After W. A. McCubbin, Pa. Dept. Agr. Bul.* **382.**)

in the recognition of yellows. The willow shoots may be distinguished from ordinary twigs or water sprouts by their greater slenderness, by the tendency to grow upright and by the tendency of their branches to grow erect rather than spreading. The willow shoots may be terminal, or they may be lateral, but they are most frequent on the larger limbs. They may appear before bearing age or may precede prematuring in bearing trees but are generally regarded as being associated with advanced stages of the disease. These willow shoots frequently continue growth in the fall after the normal shoots have stopped. It has been noted that in trees affected with yellows, both leaf and blossom buds start into growth

earlier in the spring than do normal trees. In diseased trees, a single branch will sometimes be in full bloom when the fruit buds on the remainder of the tree are unopened. The clusters of broom shoots are frequently quite conspicuous in trees in their winter condition and serve as a means of recognizing yellows when the trees are dormant.

3. *Foliage Changes.*—The foliage changes are quite characteristic and have suggested the common name applied to the disease. The shoot axes are short, the leaves generally pale green or yellow, reduced in size, being narrower than the normal and more or less drooping or rolled or curled. While these foliage changes are characteristic of yellows, they are not diagnostic, since very similar responses may result from the work of borers, drought injury, mechanical injury, malnutrition, winter injury or the related trouble known as "little peach." The sickly yellow color is not always evident in yellows, as diseased trees abundantly supplied with available nitrogen may actually show a rich-green color.

The most constant and reliable symptoms of an early stage of yellows consists of a characteristic drooping of the leaves toward the branches and trunk of the tree, combined with a slight rolling or curling of the tips toward the petioles and sometimes a rolling inward of the margins as well. Such leaves lose their flexibility and are frequently smaller than normal leaves. They vary from a light green in color to a yellowish, mottled green. Such leaves often show a lighter and yellower green than normal leaves and a reddish margin. This appearance of the foliage is quite distinct from the drooping and flaccid condition of leaves upon trees suffering from drought. Young trees affected with this disease are usually somewhat checked in growth and assume a more upright and less spreading habit than normal trees (Blake, Cook and Connors, 1921).

The rolling of leaves from the margin inward may make them more or less tubular, and in some cases this is the only warning of yellows on young trees, or the first indication of the disease on older trees.

Trees once infected with yellows never recover. Affected trees may die the second year after the first evident symptoms of the trouble, but the progress of the disease is frequently slow, the branches succumbing from the top downward. While it is true that affected trees

. . . will be weakened and may die within a few years after the appearance of the disease, it is also true that many diseased trees may remain alive and persist as long as many normal trees under usual conditions of growth.

Losses from Yellows.—The losses from yellows are due to reduced production as a result of the death of trees and to a shortening of the life of the orchard, since with the removal of many trees the maintenance of the healthy remnant is no longer profitable. The losses are heavy, since yellows does not become pronounced until trees are of bearing age, and then they must frequently be sacrificed before they have produced a crop. Before the practice of immediate removal of diseased trees was started,

the peach-growing industry was nearly swept out of existence in some parts of the eastern United States and in sections of the Michigan peach belt. The serious character of the disease may be illustrated by the losses in certain orchards:

Orchard 1: (1884–1887) 887 of a total of 1777, or about 50 per cent.
Orchard 2: (1881–1887) 2016 of a total of 2146, or about 94 per cent.

According to Blake *et al.* (1921):

> In a yellows and little-peach district, a loss of 1 per cent of the trees annually is low even during what might be called a quiescent period between epidemics, while a loss of from 2 to 3 per cent by infection per year is common. During so-called epidemics, the percentages may increase to 25 per cent even in orchards where the diseased trees are removed annually, as was the case at Vineland.

It is estimated that an annual loss of 3 per cent for New York, New Jersey, Pennsylvania, Delaware and Maryland would mean 450,000 trees.

Etiology.—Peach yellows has been recognized for some time as belonging to the obscure type of the "virus diseases," although it differs from many of the diseases in this class in its contagious or infectious properties. The work of Smith gave definite proof of the infectious character of the disease. He proved that it could be transmitted from diseased shoots to healthy trees by budding (1888, 1889) and that apparently healthy buds from diseased trees would also communicate the disease to the trees on which they were inserted. The smallest amount of bud tissue that could be used and form a union was sufficient to communicate the disease. The time of incubation following an inoculation by budding has been shown to vary with the virulence of the disease in the tree from which buds are taken and also according to the portion from which they originate. In the most rapid progress of the disease, buds inserted in August showed characteristic yellow shoots immediately the following spring (Blake *et al.*, 1921). June budding did not appear to advance the appearance of the disease; and when buds were taken from mild or early cases of yellows, the inception and progress of the disease were somewhat slower, the first symptoms sometimes appearing late in the season following the inoculation.

For many years, it has been known that yellows will spread from diseased trees to adjacent healthy trees. The disease may appear on isolated trees scattered throughout an orchard, apparently as if brought from a distance by some carrier, or centers of infection from which the disease radiates may be evident, with all or only part of the trees within a circular area infected. The method of transmission under natural field conditions has never been determined, but McCubbin (1928) thinks that there must be another host and an insect vector. At the present time, there is no positive evidence that the disease can be transmitted by the pruning tools, pollen from yellows trees or the feeding of

sucking insects, as has been proved in so many of the virus diseases. Inoculations of healthy trees with juice from parts of diseased trees have likewise failed to communicate the disease. The statement has been made by some that yellows is spread by the use of pits from diseased trees, but seedlings grown from such pits have remained healthy during the few years under observation, according to tests made in New Jersey (Blake *et al.*, 1921) and Pennsylvania (McCubbin, 1924). Replants set in the place of diseased trees have shown no more tendency to contract yellows than the general trees of a planting, thus indicating that the soil does not carry the disease.

Various observations point to the fact that yellows may remain latent for a long period following an inoculation. Even in infection by budding from trees "showing slight symptoms of the disease, it is quite likely that nursery trees may be produced from them which will not show advanced stages of yellows for three or four seasons after planting." Masking of symptoms or a long period of incubation may be responsible for many of the negative results which have been obtained by the various methods of inoculation.

Previous to the definite recognition of yellows as a disease of the virus type, many different theories were advanced to explain its origin. Almost the entire category of possibilities has been drawn upon: (1) biological factors, including bacterial, fungous or insect parasites; (2) unfavorable climatic factors, especially winter injury; (3) unfavorable soil factors, especially deficiencies in essential elements; and (4) injurious cultural practices or improper management, such as crowding of trees, excessive cultivation or overbearing. None of these factors has other than a modifying influence on the progress of the disease, which must be viewed as due to some infective principle transmitted from diseased to healthy trees.

Whatever this infective agent may be, it is certain that it produces in the nutrition of the tree a profound disturbance, which is expressed by the symptoms recorded. It has recently been pointed out (Cook, 1922) that the translocation of starch is greatly retarded in the leaves and twigs of trees affected with yellows, which would indicate an interference with normal enzyme activity. From the foliage symptoms, it would appear that not only is translocation of carbohydrate food affected but the photosynthetic process must be materially lowered.

Varietal Susceptibility.—The yellows is a specific virus trouble which affects primarily the peach, although it is said to occur on nectarines, almonds and apricots, while a very similar trouble has been reported as affecting plums. Yellows is a common name applied to diseases affecting various other plants, but the use of the term is no indication of any supposed identity of the troubles, indicating only a similarity of certain symptoms. The same variety growing under slightly different conditions

will show marked differences in the severity of the infection; but in a comparison of varieties, but slight differences can be noted in their resistance to either yellows or little peach. Seedling peaches are said to be more sensitive to yellows than budded trees.

Control.—The first fact to be recognized in the control of yellows is that a tree once affected has never been known to recover and that such diseased trees are a constant menace to the health of surrounding healthy ones. The prompt removal of trees just as soon as yellows is evident is the only method which will make possible the maintenance of peach orchards in localities in which the disease prevails. Loss will invariably be experienced; hence the problem of the grower is to hold this down to a minimum and thus prolong the profitable production period. The belief prevails that yellows spreads as long as the tree is in leaf but that spread during the dormant period is doubtful. Inspection of the orchard should be made when the diagnostic symptoms of the disease are evident, and every affected tree marked for removal. These should be removed completely without delay, or at least all the branches should be removed and piled up against the trunk to dry, after which the stump may be dug or pulled. The entire lot should be burned, or the trimmings burned and the larger pieces sawed up for fuel. If the trunk or stump is left temporarily, care should be taken to keep any sprouts removed, since these are considered as great a source of infection as the foliage of the tree in its original diseased state.

Trees showing symptoms of yellows on a single branch cannot be saved by cutting off the affected parts, since the disease is systemic, and apparently healthy parts are already harboring the infective principle. Complete dehorning of an affected tree is of no avail and should never be attempted as a means of saving it. Reported "cures" for yellows are all fakes, while applications of fertilizers are without any effect except temporarily to stimulate growth or to make the trees greener for a time. Trees should, however, be given the best of care, and cultural methods approved for the locality should be practiced, as well-nourished, vigorous trees suffer less from yellows than the neglected orchard. While it is not certain that yellows is carried by nursery stock, it is advisable in starting an orchard to avoid weak, sickly looking trees, as they are dear at any price. The losses from yellows will be reduced to a minimum if the following conditions are met: (1) good nursery stock; (2) favorable, well-drained orchard sites; (3) good cultural practices; (4) regulated and careful inspections for the presence of yellows; and (5) the immediate removal of all trees showing signs of the disease.

While the grower should be his own inspector and be constantly on the watch for the first evidences of yellows, it is important to note that there are several times during the growing season when systematic inspection will give the best results. These are as follows: (1) at blossom-

ing, when the symptom of early blossoming may be looked for; (2) about July 1 for foliage and twig symptoms; (3) 10 days before the normal ripening period for prematures, the most reliable evidence of yellows; and (4) in late summer, when foliage symptoms may become more pronounced or new willow twigs may develop. In some states, regular state inspection (beginning July 1 and Sept. 1) of all commercial orchards is in operation to supplement the efforts of the owners. Such inspection was instituted in Pennsylvania in 1921, when 4.5 per cent of the trees were found affected. The next year, yellows had dropped to 2.5 per cent; and in 1923, only 2.21 per cent were recorded. Practically all states have general horticultural laws which would make possible the compulsory destruction of trees affected with yellows, and some have special legislative enactments designed to bring about general and effective eradication. In the practical enforcement of eradication measures, it may be assumed that an orchard more than 300 yards distant from an infested orchard will be safe from anything except isolated cases. The close infested orchard is a decided menace to a healthy one, and the healthy orchard should be protected.

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LITTLE PEACH

This disease of the peach is characterized by delayed ripening, undersized fruit, yellowish drooping leaves and invariably ends in the death of the affected tree.

History and Geographic Distribution.—Little peach was reported by Smith (1898) as prevalent in Michigan, but it had caused serious losses in that state prior to 1893. There is a belief that it existed in New York also at about the same time, although the evidence is not positive. Like yellows, its origin is unknown, but it has been suggested that it was imported with Japanese plums. By 1910, the disease was widely distributed over the state of New Jersey and is known to have been prevalent for several years previous to that time. Special studies of little peach were reported from Ontario in 1912, from New York in 1914 while recent work has appeared from New Jersey (Blake *et al.*, 1921) and from Pennsylvania (McCubbin, 1924). It appears to be prevalent throughout much of the same territory as peach yellows, although the two diseases are believed to be entirely distinct. Little peach has been especially serious in portions of Michigan, New Jersey and Pennsylvania and in some cases has been more destructive than yellows.

Symptoms and Effects.—The differentiation of little peach from yellows is difficult except in bearing trees, but in this case the characters are unmistakable. The symptoms may be grouped as follows:

1. *Fruit Effects.*—Undersize, the character which has suggested the common name, and delayed maturity are the most reliable earmarks of the disease.

Apparently the lateness of ripening and the decrease in size differ according to the severity or stage of the disease, varying from nearly normal-sized fruit ripening almost on time to very small fruit maturing 10 days or more later than normal (McCubbin, 1924).

The flavor of such fruit when ripe is inferior, and in some cases it is decidedly insipid and watery, but it may be as highly colored as normal fruit. The flesh appears to be somewhat "stringy," at least with early clingstone varieties (Blake, 1910).

The blotching of the skin and the discoloration of the flesh so characteristic of yellows are absent. The pits of affected fruit are smaller than normal, more or less shriveled and generally fail to sprout. In certain varieties at least, the affected fruits vary from the normal form, being more or less flattened and somewhat rectangular.

A condition known as peach "buttons" should not be confused with little peach. Certain varieties may produce some small fruits or "buttons" which never grow to be much larger than a hickory nut and cling to the tree throughout the season. These have been attributed to either cold or severe weather or to partial or complete impotency of the pollen.

2. *Vegetative Effects.*—In young trees or in older trees that are not bearing, foliage changes are characteristic of the disease and offer the only means of recognition; while in bearing trees, the same foliage symptoms may be in evidence in addition to the abnormal fruiting

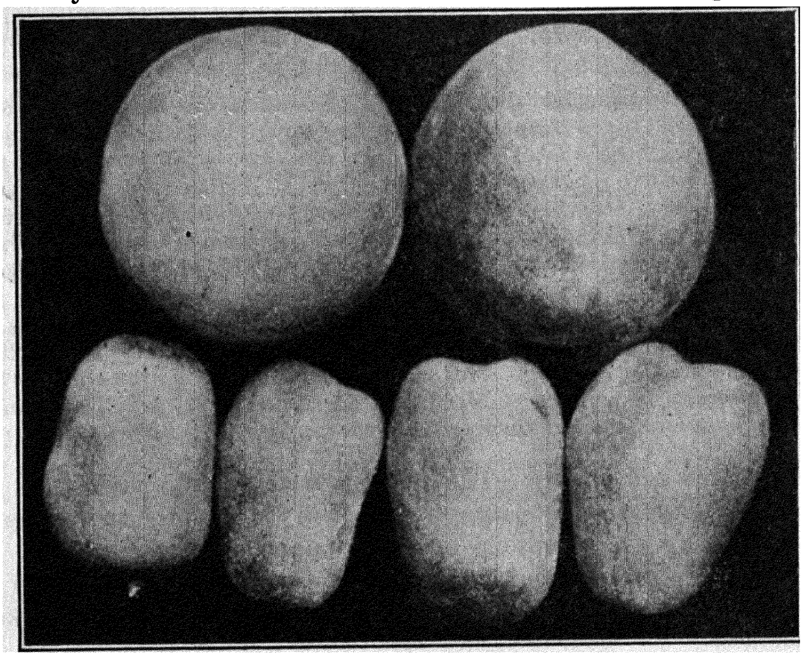

FIG. 75.—**Normal and little-peach** specimens of Greensboro. (*From N. J. Agr. Exp. Sta. Bul.* **356.**)

The foliage may vary from a lighter green than normal trees to a decidedly yellowish green, but the leaves at the base and through the center of the tree will have a rolled and a drooped appearance. When the tree is severely checked, all the leaves will have this appearance. Trees in certain stages of yellows may also have a similar appearance; and unless the trees are bearing fruit, it is almost impossible to distinguish any difference. However, the appearance of the leaves once carefully observed is not likely to be confused with anything except yellows or little peach, and this is sufficient from the practical orchard standpoint (Blake, 1910).

Trees affected with little peach never develop the willow or broom shoots which are so diagnostic of yellows. Some observations, however, have shown that young trees of certain varieties at least sometimes show

a more upright growth with the twigs shorter and more numerous than in healthy trees.

Trees may sometimes be affected with both yellows and little peach, some branches showing the prematures of yellows, some normal fruits and others typical little-peach fruits. Mixed infections do not appear, however, to be very common.

Trees affected with little peach never recover. The progress of the disease may be variable, but there is a general decline in vigor. Affected trees may continue to bear fruit, but it becomes smaller and poorer, the branches die back and the tree finally succumbs. The quality of the soil, culture practices or the use of fertilizers may modify the rate of decline, but affected trees do not last more than 3 or 4 years. During most of this period of decline, their retention in the orchard would be unprofitable because of partial crops of poor quality, even though they were not a menace to other healthy trees. In some localities, little peach is reported to be more prevalent than yellows, and the decline of affected trees more rapid.

Etiology.—Little peach has been studied much less than yellows, and but little real progress concerning its cause has been made. It is contagious, the same as yellows, and can be transmitted from diseased to healthy trees by budding. There is no evidence that little peach and yellows are anything but distinct and independent diseases. Most of the facts concerning the etiology of yellows would apply equally well to little peach. There is a deep-seated interference with the nutrition of the diseased tree which is visibly expressed by the recorded symptoms, and there is the same interference with the translocation of carbohydrates as in yellows. It is difficult to explain how this accumulation of starch should accompany such diverse symptoms as are shown by the fruits in the two diseases—enlarged fruits and early maturity in yellows and undersized fruit and delayed ripening in little peach.

Control.—The recommendations made for the control of yellows apply equally well to the control of little peach. Since diseased trees cannot be cured by any known treatment and are a constant menace to the health of surrounding trees, immediate removal is the only method of control. Inspection of peach orchards for yellows and little peach should go hand in hand with the understanding that the prompt use of the ax is equally important in the two diseases.

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See also BLAKE, 1910; CAESAR, 1912; ATWOOD, 1914; BLAKE, COOK and CONNORS, 1921; COOK, 1921, 1922; and MCCUBBIN, 1924, 1928, under Yellows references.

PEACH ROSETTE

This virus disease of the peach is known chiefly in Georgia, South Carolina, Alabama, Missouri and Tennessee, but a few cases have been reported from Kansas, Illinois, Arkansas, Oklahoma, West Virginia, Mississippi and Florida. What appears to be the same disease has recently been reported from Italy (Ferraris, 1928).

In general, the means of spread and control are similar to yellows, but its progress is more rapid. The leaf buds of affected branches

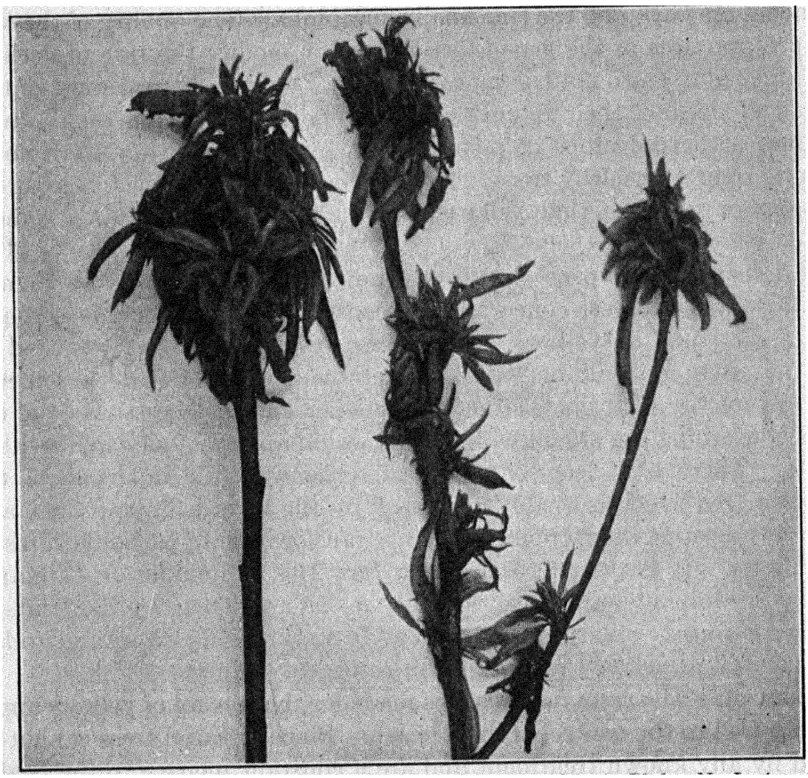

FIG. 76.—Twigs from peaches affected by rosette. (*After M. A. Blake, N. J. Agr. Exp. Sta. Bul.* **356.**)

develop compact tufts or rosettes, which may contain several hundred small leaves on axes not more than 2 or 3 inches long. A tree suffering from a general infection will always succumb during the following autumn or winter, but partial infections may occur. In such cases, the diseased limb dies, while the remainder shows the trouble the next season.

The prevailing color of the foliage is yellowish green or olivaceous. The older leaves at the base of the tufts are largest and frequently grow to a length of several inches but have inrolled margins and a peculiar stiff appearance due to the fact that they are straighter than healthy leaves. These outer leaves

turn yellow in early summer and drop as readily as though it were autumn, while the inner leaves of the rosette are still green and delicate. The compact bunching of the leaves is very conspicuous and makes the trees look quite unlike those affected by yellows. Where a tree is attacked in all parts, it matures no fruits (Smith, 1894).

Rosette was first proved by Smith (1891) to be infectious by buds or grafts, and more recent infection studies have been reported by McClintock (1923, 1931). Marianna plum is listed as immune, and the successful transfer of the disease is reported to two varieties of apricots, two of cultivated plum, one wild plum, one cherry, the sand cherry and two varieties of apricots by means of infected buds. On apricots and plums, mottling similar to mosaic followed the insertion of buds from rosetted trees, in addition to somewhat modified rosettes of leaves. The most likely insect vector, the peach aphid (*Anuraphis persicæ niger*) failed to transmit the disease, although repeated trials were made. Because of the mottling on some species, McClintock lists the disease as an "infectious mosaic."

A suggested relation of the yellows of *Erigeron canadensis* to rosette was not substantiated by a comprehensive series of cross-inoculation tests (McClintock, 1931). The yellows of Erigeron is caused by the same virus as aster yellows.

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WHEAT MOSAIC OR ROSETTE

This virus disease of wheat is of special interest because it represents a rather unique type, the only one known to be transmitted through the soil or by some agent operating within the soil.

History and Geographic Distribution.—This disease was first reported from Illinois by Lyman in 1919 under the name of "take-all," and at that time it was thought to be identical with the true take-all disease caused by *Ophiobolus graminis*, known in Europe and Australia but not at that time reported for America. Later investigations (McKinney, 1923) showed that the rosette disease of wheat differs from take-all in symptoms and host relations. The successive stages in the progress of our knowledge are reflected in the common names applied to the disease: take-all, so-called "take-all," rosette disease and mosaic disease. The virus nature of the disease was first suggested by McKinney (1923), who also reported that the causal agent persisted in the soil, causing the recurrence of the disease. The studies on the soil relationship of the disease were extended by Webb (1927, 1928). Since 1925, the disease has been recognized as a mosaic and the rosetted condition as but one of the symptoms, appearing only on certain varieties (McKinney, 1930). Positive con-

firmation of the virus character of the disease was first obtained by a low percentage of successful transfers by juice inoculations (McKinney, 1925*b*).

Wheat mosaic has occurred principally in a rather restricted area in Illinois and Indiana. Either the same or a related trouble has been noticed in very limited amounts in Nebraska, Maryland, Kansas and Washington. The mosaic of wheat studied in Egypt appears to be distinct from the American disease (Melchers, 1931).

Symptoms and Effects.—Under field conditions, the disease is most evident in rather sharply defined spots, although scattered individual diseased plants may occur. The marked characteristics of diseased plants are (1) dwarfing; (2) darker than normal-green color of the fall leaves; (3) dying of outer leaves and fall tillers; (4) excessive development of spring tillers, giving the condition which suggested the name rosette; (5) more or less streaking or striping with yellow, giving a mottle or mosaic; and (6) in the more extreme cases, the complete killing of plants before reaching maturity. Not all of the symptoms will appear in every case of the disease, the rosette condition appearing independent of mottling, or the mosaic mottling appearing independent of the rosette. Two different types of mottling have been recognized: (1) the *green type*, showing either "a light-green pattern on a normal-green background" or with increase of the light-green areas appearing like a dark-green pattern on a light-green background; and (2) the *yellow type*, showing light-yellow patterns or irregular strips (McKinney, 1925*a*).

The damage from the disease is due to a complete killing of plants and a dwarfing of others which either fail to fruit or produce a few undersized, weak culms. In the area of principal infestations, entire fields have been plowed up, while others allowed to mature have shown up to 20 to 40 per cent reduction in yield.

Etiology.—The virus nature of this wheat disease has been established by (1) the successful transmission of the disease by the growth of susceptible varieties in infested soil; and (2) by successful juice inoculations from diseased to healthy plants. By successive transfers from green mosaic, this type has been intensified, and the yellow type has been intensified by successive transfers from plants affected with the yellow type. X-bodies have been noted in the typical green mosaic, but in yellow mosaic they seem to be either few in number or absent.

The Currell variety, selections of several other wheat varieties and Red Winter spelt develop yellow mosaic to some extent in the spring when the seed is planted out of doors in virus-infested soil in the autumn, whereas certain other varieties of wheat (Harvest Queen) when grown simultaneously in the same soil and in adjacent rows develop green mosaic, become dwarfed, producing a condition which has been termed rosette and show only occasional cases of yellow mottling, striping or streaking.

As a result of numerous inoculations, McKinney (1931) concludes:

> It is evident also that rosette is not associated with yellow mosaic on Harvest Queen wheat, whereas it is associated with green mosaic on this variety. The nature of the association between rosette and green mosaic must be studied farther. A single virus may cause both expressions, or more than one virus may be involved.

No insect vectors are known, neither is the disease transmitted through the seed.

The expression of the disease is influenced by temperature conditions. In tests at constant temperatures of 10, 16, 23 and 30°C., the disease occurred at the two low temperatures only. The leaf mottling and rosette of the Harvest Queen developed best at 16°C., while the leaf mottling of Currell was about equal at each of lower temperatures. No mottling or rosette occurred in either variety at the two higher temperatures. A high moisture content of the soil has favored the occurrence of both rosette and mottling, but both have been inhibited by low moisture of the soil. Infection and onset of the disease are influenced by the age of seedlings when exposed to contaminated soil, those 4 weeks old appearing more susceptible than those either younger or older (Webb, 1927).

Host Relations.—Wheat-mosaic symptoms are expressed only in winter varieties, the characteristic features showing best in fall seedings, but the disease will appear in mild form in winter varieties sown early in spring if the temperatures are sufficiently low. The disease has appeared under natural field conditions on various varieties of winter wheat and also on winter rye. Infections have been produced in the following cereals, all of the tribe Hordeæ: common and club wheats, Poulard wheat, durum wheat, emmer, spelt, Polish wheat, einkorn, common barley and rye.

A large number of winter wheats have been listed for resistance, some of the most susceptible such as Harvest Queen showing 95 to 100 per cent infection, others only traces of infection, while others appeared to be entirely immune.

Control.—The disease in wheat can be controlled by the use of either resistant or immune varieties. Fields in which the soil is contaminated should not be sown to such susceptible varieties as "Harvest Queen, Missouri Bluestem, Nigger, Penquite, Brunswick or certain selections of Fultz, Indiana Swamp and Illini Chief" (McKinney, 1925*a*). The mottle symptom is very pronounced on Currell, but it does not develop the rosette symptom.

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CURLY TOP

This virus disease of the beet and numerous other hosts has been referred to on beets at various times and places as the "California beet disease," "western blight," "blight," "whiskered beets," "hairy root," and "curly leaf" but is now generally known as "curly top." The last name has seemed most appropriate, because rolling and curling of the leaves are the most striking effect of the disease.

History and Geographic Distribution.—The first serious outbreak of curly top of definite record occurred in California in 1899, but it was more severe the following year. The disease had previously caused concern to the sugar-beet industry. The first scientific work on it began about this time and has increased in volume up to the present time. Townsend described the disease in 1902 and in 1908 discussed the various theories as to the cause but arrived at no definite conclusion as to the etiological agent. The study of Smith in connection with the California epiphytotic of 1905 led to the supposition that the disease might be of a nature similar to tobacco mosaic or aster yellows. Ball (1906) first directed attention to the beet leaf hopper as causally related and soon (1909) presented convincing evidence that this insect is responsible for curly top. This relationship of the leaf hopper to the transmission of the disease was confirmed and elaborated by the work of Shaw (1910), Smith and Boncquet (1915) and by Ball (1917). The years following have yielded a continuous output of researches on the disease by Carsner and his associates from the Federal Laboratory at Riverside, Cal., and from Severin of the California station and others. Since the discovery of the causal relation of the curly-top virus to the western blight of the tomato (McKay and Dykstra, 1927), the host range of the disease has been rapidly extended (Severin 1928, 1929; Dana, 1932), and the importance of the disease on crops other than beets emphasized.

The curly top is primarily a disease of arid or semiarid portions of western North America, its distribution coinciding with the range of its vector the beet leaf hopper (*Eutettix tenellus* Baker). The regions of principal severity are west of the Rocky Mountains, but there are sporadic cases in the plains country east of the mountains. The same trouble or one very similar has been reported from the Argentine (Fawcett, 1925, 1927), but another vector is prevalent.

Symptoms and Effects.—The following are the recognized responses of the beet foliage to infection: (1) leaf curling; (2) blister-like elevations on the leaves; (3) transparent venation of the innermost or youngest leaves; (4) wart- or knot-like swellings on the veins of the lower surface; (5) the exudation of a viscid sweetish liquid from the petiole, midrib or veins, clear at first but later dark and drying to form a brown crust; (6) yellowing and blighting of the leaves; and (7) a retardation of growth. The earliest symptom of the disease is an inward rolling of the lower and outer margin of the youngest leaves or sometimes an outward rolling of the margins or even a combination of the two types of curling.

The effects on the beet root are (1) the production of an increased number of lateral rootlets, giving the condition suggesting the common names of "hairy or woolly root" or "whiskered beets"; (2) a necrosis of the phloëm extending throughout the vascular system and evident in the cross-section of the root as dark concentric rings (Fig. 75); (3) a reduction in the size of the roots and a reduced sugar content, in the moderate degrees of infection not involving killing; and (4) shriveled, dead or rotted roots as an accompaniment of severe blighting and sun scorching of the foliage.

The effects on seed beets are (1) the production of "dead heads" or of blighted or dwarfed seed stalks and (2) reduced yields of seed of poor viability. Shaw (1910) reports that healthy seed beets produced twenty-eight times the quantity of seed obtained from infected roots.

The degree of injury in curly top is influenced by the time of infection. Young seedlings may be killed outright; but if the infection is delayed, only the newly formed leaves will exhibit the symptoms described. Infections occurring late in the season may not be evident, but such beets if used for stechlings would develop only diseased seed stalks.

Curly top on garden vegetables such as tomato, bean, squash, etc., often kills the plants in the seedling stage or in older plants causes the development of dwarfed and crippled, chlorotic individuals which may die prematurely. The response of tomato plants is very characteristic, including retarded growth, upward rolling of the leaflets, more or less chlorosis, a rigidity or harshness of the foliage, premature ripening of the fruit and frequently a death of the plant before killing frosts. Crop losses from curly top may vary from slight injury to complete failure.

Etiology.—Curly top is a virus disease which depends upon the beet leafhopper (*Eutettix tenellus* Baker), for its dissemination under natural field conditions. This relationship of the leafhopper to the disease has

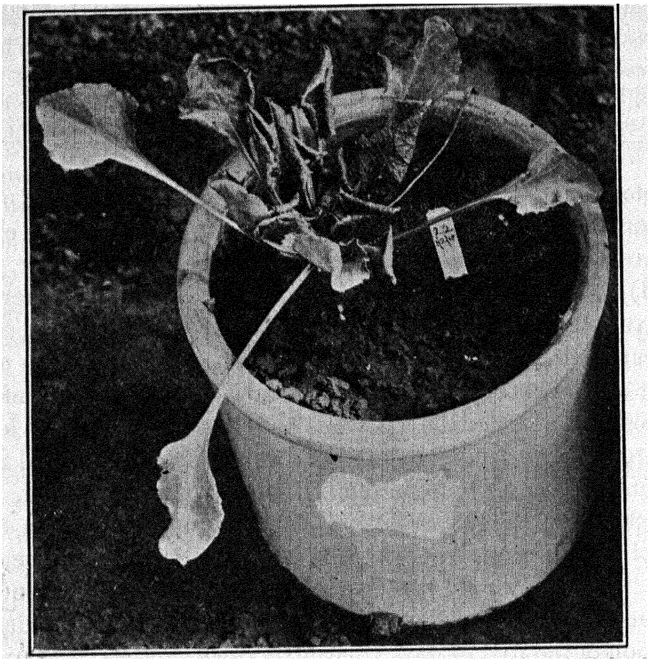

FIG. 77.—Appearance of severe curly top of young sugar beet. (*After Eubanks Carsner.*)

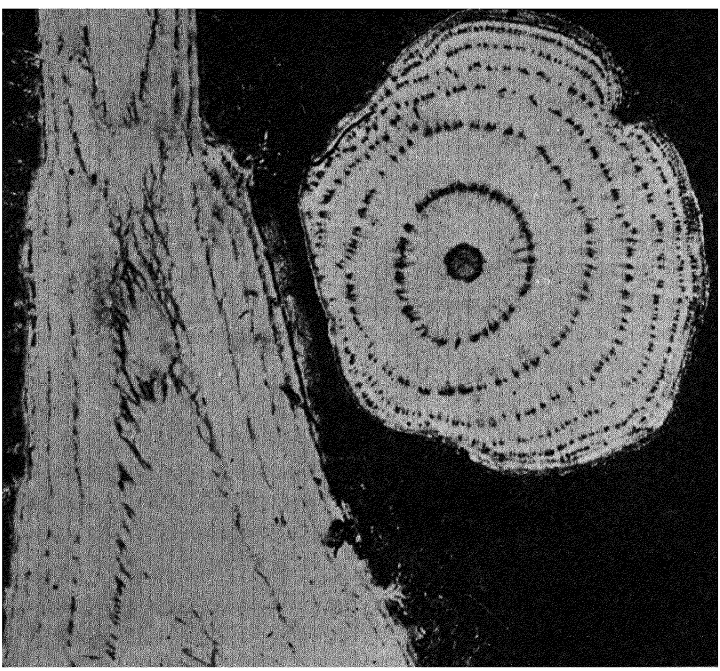

FIG. 78.—Phloëm necrosis in sugar beet affected with curly top. (*After Carsner and Stahl, Jour. Agr. Res.* **28.**)

been repeatedly demonstrated since the first work of Ball (1906, 1909). The early reports concerning the causal relation of bacteria to the disease (Smith and Boncquet, 1915) were not substantiated. The severity of the disease in any environment will depend upon the abundance of the leafhoppers and the time of their appearance. Only leafhoppers that have fed on a wild or cultivated host harboring the disease become viruliferous, *i.e.*, are able to transmit the disease when they feed on healthy suscepts. Newly hatched nymphs cannot transmit the disease until they have fed on diseased foliage; and in all cases, a period of incubation in the body of the hopper is necessary before infection will result. This "varies within quite wide limits, from the so-called normal incubation period, or the period when at least 50 per cent of infection should be obtained, down to a single infection in as short a time as 1 hour" (Swezy, 1930). Infection may be transmitted by a single hopper, but the action of numbers, or mass action, is very much more effective. The incubation within the host varies from 7 to 14 days, with occasionally an earlier onset of symptoms (4 days).

The onset of the disease in any season may depend on the holding over of a few viruliferous adults which feed on the newly planted crop and start the infection, or the spring broods may obtain their virus from infected wild plants in their natural breeding areas or even from cultivated symptomless carriers.

The artificial transmission of curly top is much more difficult than in many other virus diseases. Early efforts gave negative results, but more recently Severin (1924) secured a low percentage of infection by pressing the juice of leaves and roots of curly-leaf beets into the crown of healthy plants. The causative agent was shown to be generally distributed in both foliage and roots. The disease has been transmitted by leafhoppers which have fed on (1) a suspension of crushed viruliferous leafhoppers; (2) diseased beet juice in 1 per cent aqueous solution of sucrose; and (3) a solution on which viruliferous hoppers had previously fed (Carter, 1928). Until recently, *Eutettix tenellus* was supposed to be the only vector, but this specificity has been denied by the discovery that another leafhopper, *Agallia sticticollis*, is a common vector in the Argentine (Fawcett, 1927).

Host Relations.—The curly-top virus affects many species of plants, both wild and cultivated. The disease has been recorded in either mild or severe form as natural infections on the following cultivated species: garden and sugar beets, mangels, Swiss chard and spinach (Chenopodiaceæ); common and Lima beans, cowpeas and alfalfa (Leguminosæ); pumpkins, squash, watermelon, cucumber, muskmelon and cantaloupe (Cucurbitaceæ); potato, tomato and pepper (Solanaceæ); horse-radish, radish, cabbage and turnip (Brassicaceæ); parsley (Umbelliferæ) and various cultivated ornamentals including Zinnia, African daisy, etc. In

addition, successful artificial inoculations have been made on many other cultivated species.

In addition to the cultivated hosts, many wild plants are known to be infected under natural conditions, and others have been shown to be susceptible. It is certain that some of these weed hosts are sources of the virus for the spring crops of leafhoppers. Some of these important weed hosts are various species of Atriplex, certain species of Chenopodium, Russian thistle, amaranths, ground cherry, deadly nightshade, charlock, shepherd's purse, knotweed and other species of Polygonum, dwarf mallow, cheese weed, alfilaria, or filaree and oxalis (*Oxalis stricta*).

Individual selections of sugar beets have shown varying susceptibility to curly top, and in some cases the virus has become so attenuated that beets carrying it do not show visible evidence of its presence. It has been shown that apparently healthy resistant beets may then act as symptomless carriers and yield the virus to leafhoppers that feed upon them. Resistance is an inherent character, and some selections have shown more resistance than commercial fields. Among vegetable crops, varying resistance is also shown. No varieties of tomato have shown a satisfactory resistance, but marked resistance has been found among beans, squash and pumpkins (Dana, 1932).

Control.—The high temperature, bright sunshine and low humidity of the semiarid regions offer conditions for the development of large numbers of the insect vector and the expression of diseased conditions. Little can be done in modifying the environment or reducing the numbers of leafhoppers by either artificial or biological methods. The ultimate solution of the curly-top problem lies in the selection and breeding of resistant varieties, but some relief may be obtained by cultural practices, such as time of planting (sugar beet and tomato) or providing a shade crop (tomato).

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POTATO MOSAIC

Mosaic is the general name applied to a group of the virus diseases of the potato characterized by more or less mottling of the foliage, especially when potatoes are grown under suitable conditions of moisture and temperature, while various other accompanying symptoms may be present

in the several types. The forms of mosaic are among the most important of the degeneration diseases of the potato which are responsible for the "running out" of varieties.

Potato-degeneration Diseases in General.—In addition to the mosaics and mosaic complexes, the following specific degeneration diseases of the potato may be recognized:

1. *Leaf roll,* characterized by rolling of the leaves and dwarfing, uprightness, rigidity and chlorosis (see special treatment).

2. *Spindle tuber,* a disease

> . . . characterized always by spindliness and uprightness and often by a darker green color and slight rugosity. The tubers are abnormally spindling, spindle-shaped, cylindrical and supplied with conspicuous eyes, these symptoms varying somewhat with the variety (Schultz and Folsom, 1923).

Affected plants show no mottling, a fact which makes possible a ready separation from mosaic. This disease is a most important cause of running out of potato stock in which poor tuber shape is a common character (Folsom, 1923; Werner, 1925). "Giant hill" has recently been described as a phase of spindle tuber (Gilbert, 1925). Recently it has been pointed out that unmottled curly dwarf shows symptoms under certain conditions which make it nearly impossible to separate it from spindle tuber (Goss, 1930). According to Schultz and Folsom (1925), mottled curly dwarf seems to be a combination of leaf-rolling mosaic and spindle tuber.

3. *Witches' broom,* a disease recently described by Hungerford and Dana (1924), marked by a bushy clustering of numerous slender sprouts, a character which has suggested the common name. "One of the most constant symptoms of this disease in all stages is the tendency for all buds, including those usually latent on a normal plant, to push into growth." A more upright position than normal of stems, leaves and flowers clusters gives a characteristic picture. Very marked reduction in size of the tubers is also characteristic, together with elongation of stolons and increase in number of tubers. As many as 200 tubers varying in size from peas to walnuts may be produced in a single hill. Transmissible to tobacco and tomato by grafting (see virus diseases, pp. 321–322).

4. *Psyllid yellows,* a disease prevalent from Colorado westward, characterized by marked stunting of the plant; upward rolling of the basal portion of young leaves, becoming light pinkish yellow to purple in some varieties; and yellowing, upward rolling and death of older leaves. Axillary buds may form aerial tubers, thick shoots or rosettes of small leaves. The vector is the potato or tomato psyllid (*Paratrioza cockerellii*), which is capable of transmitting the disease to other solanaceous species including common peppers, tomatoes, eggplant and Jerusalem cherry (Richards, 1928; Binkley, 1930).

5. *Calico,* characterized by irregular blotches of various shades of yellow on the leaflets. Transmissible by tuber grafting and by inocula-

tion of leaves with unfiltered juice. Insect vectors not yet determined, but field spread is indicated (Hungerford, 1922; Porter, 1931).

6. *Phloëm parenchyma necrosis* of the tuber (pseudo-net necrosis), characterized by spot-like rather than blotchy necrotic areas in the tubers but showing no foliage symptoms. The disease is tuber perpetuated and is transmissible by aphids and by juice inoculations (Quanjer, Thung and Elze, 1929; Kerling, 1929; Quanjer, 1931). This appears to be identical with "vererbliche Eisenfleckigkeit" of Fruwirth.

7. *Concentric necrosis* of the tuber, characterized by "necrotic spots in the storage parenchyma demonstrating themselves on the cut surface as concentric brown rings arising from some point on the skin, often a lenticel." It is not tuber transmitted, but infection is thought to take place through the soil by way of the lenticels. In this type the virus nature does not appear to be positively established. This is the "Kringerigheid" of Holland, the "Propfenbildung" and in part the "Eisenfleckigkeit" of Germany and may be identical with some of the American and English "internal brown spot," "sprain" or "internal rust spot" (Atanasoff, 1926; Quanjer, 1926, 1931).

8. *Curly top* of beet and other hosts has been reported on potato from natural field infections and has been produced experimentally on eight or more varieties. The affected plants are stunted, with yellowish, inward-rolled leaflets and in the greenhouse a downward curving of the petioles. Dwarfed axillary shoots were formed in the advanced stage of the infection, and the plants finally turned yellow and died (Severin, 1929).

History and Geographic Distribution of Potato Mosaic.—Although the mosaic of the potato has been recognized as a specific disease only during recent years, it is not to be understood that it is a new disease that made a sudden appearance. It has undoubtedly been active in potato fields ever since the so-called "running out" of varieties has been recognized. It seems probable that Johnson (1847) observed a form of mosaic, since the trouble which he described showed characters suggesting that disease, especially the following:

The upper surface of the leaves is not so smooth as is usual in the case with potato leaves but rough, wrinkled or curled. The leaves are far more sessile than usual, and are not of a uniform brownish or dark-green color, but spotted. Mosaics undoubtedly formed a part of the symptomatic complex included in the "Kräuselkrankheiten" or "curl" of the Germans.

Potato mosaic was first found by Orton "in 1911 in a field in Giessen, Germany, where it was not uncommon, especially on some varieties" (1914). It was present in Maine, the following year, especially on the Green Mountain, and from the fact that the disease affected up to 100 per cent of the hills in some of these fields it would seem that it must have been present for some time previous. Wortley (1915) reported mosaic as seriously affecting the Bliss Triumph in Bermuda and on Long Island. Two years later, Murphy (1917) recognized it as causing a large and steady drain on the profits of potato raising in the Maritime Provinces of Canada in which the Green Mountain variety is largely grown and reported its occurrence in western Canada also. It was observed by the writer in Washington in 1915, but its importance was

not recognized at that time because of the masking of symptoms. By 1917 and 1918, it was reported from Maine to Oregon and from Michigan to Florida and Alabama. Quanjer writing in 1921 stated that he had learned to distinguish mosaic from leaf roll 14 years before, when he began the study of the degeneration diseases. Important contributions to the knowledge of mosaic have been made by numerous investigators in recent years, including Westerdijk (1916) and Quanjer and coworkers (1919 and later) in Holland, Murphy in Canada (1917, 1921) and Ireland (1922, 1924), Cotton in England (1922), Schultz *et al.* (1919), Schultz and Folsom (1920 and later), Johnson (1922), Goss (1924), Goss and Peltier (1924) and various other workers in the state and Federal departments in the United States and Canada and foreign countries.

Some of the more important steps in the progress of our knowledge of mosaic are: (1) the recognition of mosaic as a specific disease (Orton, 1911); (2) the proof of the hereditary or perpetuating character by transmission through the tubers (Wortley, 1915); (3) the transmission by grafting (Schultz, 1917); (4) transmission from diseased plant to healthy plant by the juice; (5) transmission by aphids (Schultz, 1919; Quanjer, 1920); (6) the recognition of several types of mosaic (Schultz and Folsom, 1920–1923; Quanjer, 1922); (7) the proof that current season infections, especially late ones, may produce no visible evidence until the tubers are planted the following spring; and (8) the proof of the masking of symptoms under conditions of high temperature and low humidity (Johnson, 1922; Goss, 1924; Goss and Peltier, 1925); and (9) recognition of the nature of the rugose mosaic complex (Smith, 1930; Valleau and Johnson, 1930; Burnett and Jones, 1931). A more complete account of early work on virus disease will be found in the following paper: ATANASOFF, D. A study into the literature on stipple streak and related diseases of the potato. *Meded. van de Landbouwhoogeschool* **26** (1): 1–52. 1922.

Symptoms and Effects.—Definite description of the symptoms of mosaic is difficult, since the name mosaic as applied to the potato represents a group of closely related troubles rather than a single specific disease. Quanjer (1921–1923) recognizes six different kinds; while Schultz and Folsom (1923) have described three distinct types, but all have the features of mottling and more or less wrinkling in common.

Mottling is a localized chlorosis consisting of spotting of the leaf blades by light-green areas, which may or may not occur in contact with the larger veins, and which vary in shape and degree of paleness. These discolored spots are punctate, elongate, circular, angular or irregular. They vary from a barely discernible fading of the green to an almost pure yellow, often in the same spot. They seldom exceed a few millimeters in any dimension and their distinctness of outline differs, usually in proportion to the degree of discoloration. They are more readily seen in diffused light than in direct sunlight (Schultz and Folsom, 1923).

In addition to mottling, various other terms are used in the description of mosaic symptoms. Either *wrinkling* or *rugosity* is an abnormal unevenness of the leaf surface due to depressions and prominences, in the former of irregular height and depth, in the latter of uniform height, but with the depressions only at the veins. This is the symptom that has suggested the common name of "Gänsehaut," which is applied to the disease in parts of Germany. Especially in the extreme types, diseased leaves may be compared to the normal foliage of Scotch kale and Savoy

cabbage. Leaves showing such characters are sometimes said to be savoyed. Either wrinkling or rugosity may have a tendency to obscure

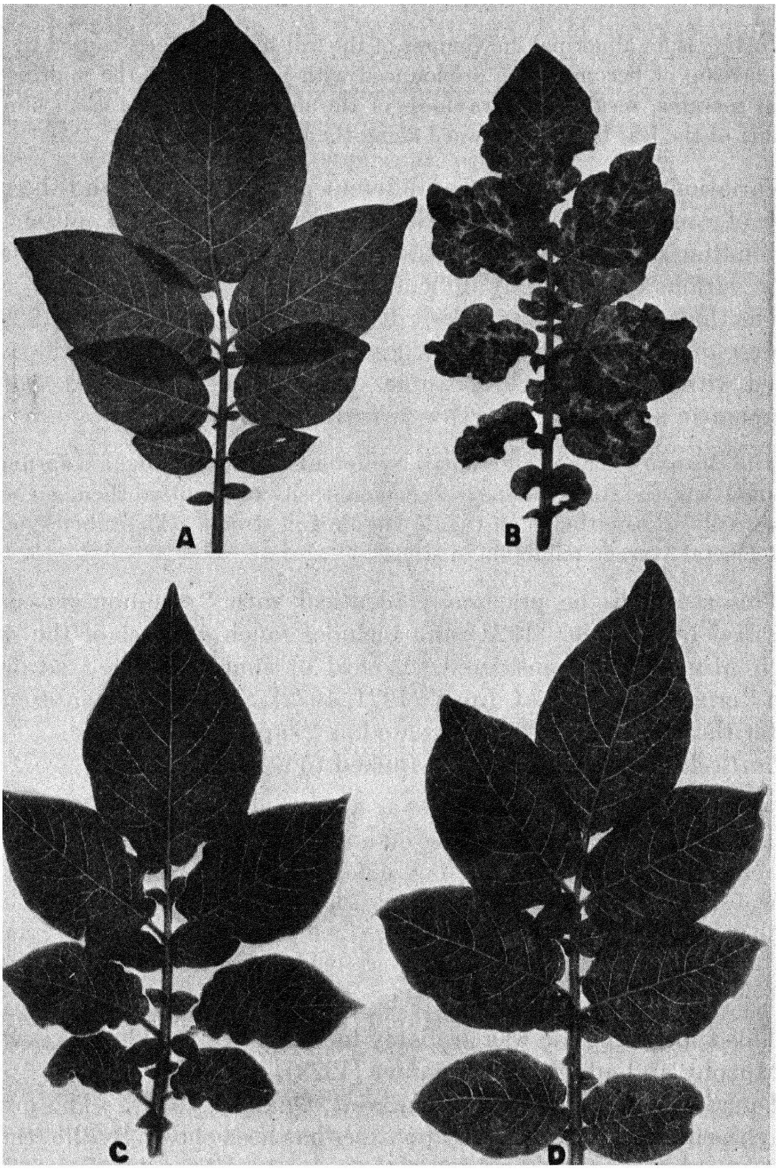

FIG. 79.—*A*, an apparently healthy leaf of Early Rose affected with latent virus; *B*, leaf of Early Rose affected with rugose mosaic; *C*, healthy leaf from potato seedling; *D*, leaf from potato-seedling plant affected with vein-banding virus. (*After Burnett & Jones, Wash. Agr. Exp. Sta. Bul.* **259.**)

the mottling, especially if this is faint. A microscopic examination of the mottled leaves will show a deviation from the normal in structure. The

chlorotic areas are thinner than the surrounding green tissue, the palisade parenchyma consisting of very short or almost cubical cells as contrasted with the much elongated palisade cells of the normal leaf (see Fig. 85).

Ruffling is an abnormal unevenness of the leaf-blade surface, caused by ridges that develop or become more pronounced with passage from the midrib to the lateral margins, resulting in waviness of the margin. *Curling* is an abnormal bending of the leaf blade downward along the main vein.

Variations in the response of different potato varieties and the occurrence of combinations of some other virus disease with a mosaic or of combination of two or more mosaics render the accurate characterization of the various types doubly difficult.

The three types of mosaic described by Schultz and Folsom (1923) will be presented first, and the forms described by other writers will be connected with these as far as possible. It must be emphasized that final judgment in some cases must await further investigations.

Mild Mosaic.—The characteristic symptoms consist of slight dwarfing, distinct mottling and some ruffling. It is more easily transmitted than leaf roll and is less easily transmitted than rugose mosaic and streak. The tuber symptoms are a general average reduction in size.

This seems to be practically identical with "common mosaic" as described by Quanjer (1923) and includes much at least of the disease which Murphy has considered "typical or simple mosaic" as distinct from "crinkle" and "leaf drop" (1921, 1922). A mosaic symptom even fainter than *mild* has been designated as "supermild" mosaic.

Leaf-rolling Mosaic.—This is applied to a

. . . symptom complex that so far has been irreducible to simpler complexes and that consists of slight dwarfing, diffused mottling, wrinkling, slight ruffling and rolling of the upper leaves. It is different from the mild mosaic in respect to the distinctness of the mottling, the presence of rolling and the effects in combination with the spindling tuber disease, and is similar to it in infectiousness. The tuber symptoms are a general average reduction in size. It is distinct from leaf roll.

This type of mosaic was probably included in "crinkle," as described by Murphy and adopted by Quanjer (1923).

Latent (*Top Necrosis or Acronecrosis*, Quanjer, 1931).—The presence of a virus in apparently healthy potatoes has been shown by the results of juice inoculations from healthy potatoes to healthy tobacco or tomato, causing a "mottle" (Johnson, 1925), sometimes very faint, at other times evident as "irregular, pale green to extremely yellow chlorotic interveinal tissue in contrast to the darker green along the veins." Necrotic symptoms may be produced in certain cases by successive transfers from originally healthy stock, or the latent of some varieties will produce necrotic

symptoms when transferred to other varieties, for example, latent in Monocraat and Roode Star will cause necrosis in Duke of York (Quanjer, 1931). A form of latent virus which differs from the common latent in its increased virulence and in the greater production of necrotic symptoms has been designated "virulent latent" (Valleau and Johnson, 1930; Burnett and Jones, 1931). Two different types of top necrosis are recognized by Quanjer, type A and type B, with at least two varieties of type A. Necrosis may occur in foliage, stem and tubers.

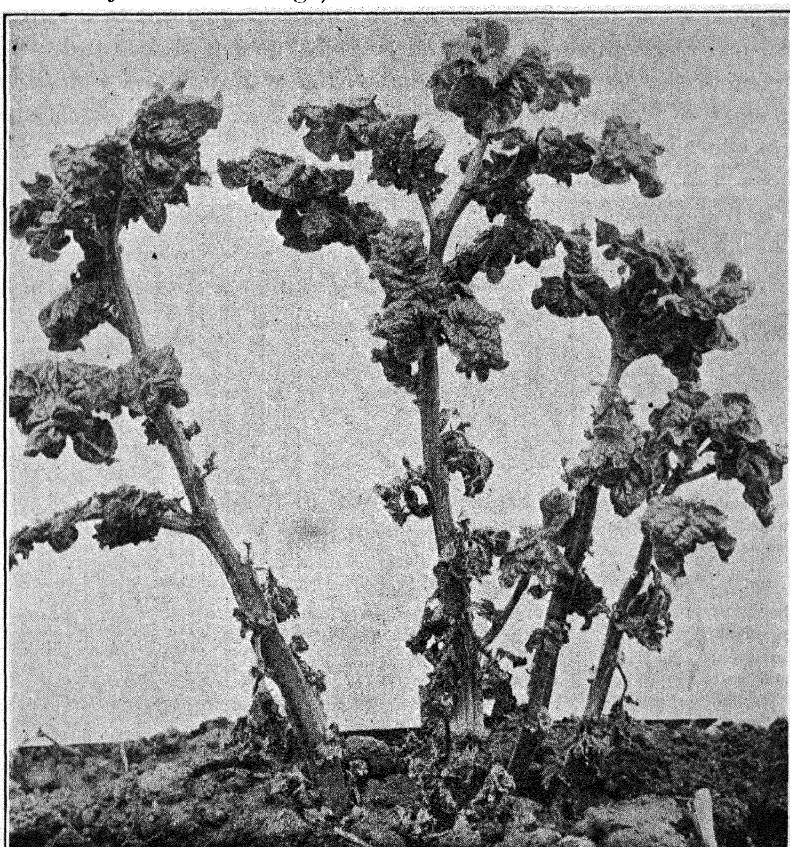

FIG. 80.—Severe form of rugose mosaic on Bliss Triumph. (*Photograph by B. F. Dana.*)

Rugose Mosaic.—This type may be differentiated from mild mosaic

> . . . by the distinct dwarfing, more chlorosis, and more diffused mottling, a more rugose type of wrinkling and a tendency to show brittleness, spotting, streaking, leaf dropping and premature death, especially when in combination with the spindling tuber disease. The tuber symptoms are a marked reduction in size (Schultz and Folsom, 1923).

The most severe symptoms from "virulent latent" may approach quite nearly to those of mild rugose mosaic. Evidence has been pre-

sented to show that rugose mosaic represents a mixed infection of at least two virus entities (Smith, 1930; Valleau and Johnson, 1930; Burnett and Jones, 1931). Inoculation tests with "latent" (x-virus of Smith) plus the tobacco "vein-banding" (y-virus of Smith) virus yielded rugose mosaic, while virulent latent plus vein banding gave severe rugose mosaic, but a more malignant rugose mosaic resulted when both virulent latent and tobacco mosaic were added with vein banding. This composite character of the rugose mosaic should explain some of the varying expressions of the complex as reported by different workers, when one takes into consideration environmental factors, variety peculiarities and varying virulence of the contributing viruses. Rugose mosaic and what appears to be some of its variations may be presented in tabular form (adapted from a chart by L. K. Jones).

Common name of disease	Authority	Symptoms and effects									
		Dwarfing	Mottling	Yellowing	Rugosity of leaves	Leaf rolling (upward)	Leaf rolling (downward)	Stem and leaf necrosis	Wavy leaf margin	Tuber necrosis	Tuber cracking
Rugose mosaic	Schultz and Folsom, 1923	x	x	x	x			x		x	
Leaf drop	Murphy, 1921	x					x	x		x	
Leaf-drop streak	Quanjer, 1923	x					x	x			
Streak	Orton, 1920	x					x	x			
Stipple streak	Atanasoff, 1922, 1925	x					x	x			
Crinkle	Murphy, 1922; Quanjer, 1923	x	x	x	x		x	x	x		
Crinkle mosaic	Schultz and Folsom, 1925	x	x		x				x		
Curly dwarf	Orton, 1914	x			x	x					x
Mosaic dwarf	Krantz and Bisby, 1921	x	x		x			x			
Russet dwarf	Hungerford, 1922	x		x				x			
Yellow dwarf	Barrus and Chupp, 1922	x		x	x	x		x		x	x
Unmottled[1] curly dwarf	Schultz and Folsom, 1923	x			x	x					x

[1] See spindle tuber for mottled curly dwarf. It seems probable that the para-crinkle and crinkle A belong to the rugose mosaic complex (Salaman, 1930; Salaman and Le Pelley, 1930).

Two other types of mosaic have also been described by Quanjer: (1) "*interveinal mosaic*," marked by pale patches between the veins, the leaf tissues directly in contact with the veins remaining green, margins often a little undulated and turned upward; and (2) *aucuba mosaic*, characterized by more or less round yellowish patches, especially evident in the upper leaf surfaces, in extreme cases occupying half of the leaf surface. The name of the latter has been suggested by its similarity to the variegation of *Aucuba japonica*.

Conflicting reports have been published as to the possibility of transferring tobacco mosaic to the potato, but Blodgett (1927) records definite

symptoms, varying with the variety. Local necrotic lesions, with no systemic infection, were recorded on Bliss Triumph, and streak-like symptoms were produced on Green Mountain.

The recognition of numerous distinct types of mosaic is, after all, not such an important thing as the recognition of symptoms of degeneration diseases in general, since they are all quite similar in their general effects and need to be held down to a minimum to insure profitable production. Mottling, spotting, streaking, blighting, leaf drop, dwarfing and early maturity either retard the photosynthetic processes or cut short the time during which this process is active. As might be expected, the interference with carbohydrate manufacture causes more or less reduction in the size of the tubers, and in some cases quality is also impaired. In

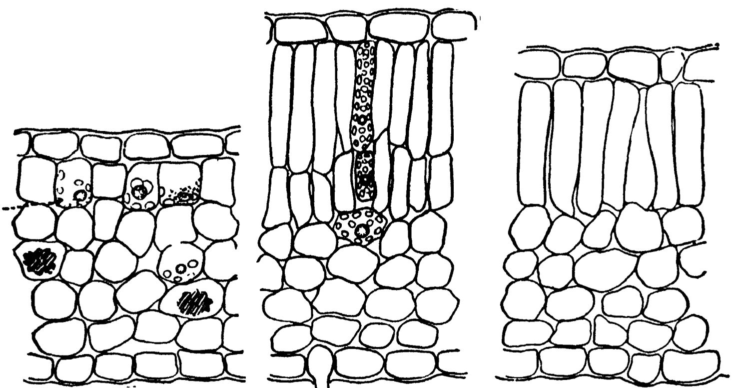

FIG. 81.—Diagrammatic cross-sections of tobacco leaves, showing effect of mosaic. *A*, section through hypoplastic area of tobacco leaf; *B*, section through dark-green area, showing hypertrophy of palisade tissue; *C*, section through ordinary dark-green tissue. (*After B. T. Dickson, Macdonald Col. Tech. Bul.* **2**.)

many cases, mosaics cause no evident tuber effects except reduction in size, there being no external or internal characters by which the disease can be identified. Cracking and splitting of tubers have been noted for streak, in curly dwarf as described by Orton, in leaf roll, in yellow dwarf as studied by Barrus and Chupp (1922) and in unmottled curly dwarf by Schultz and Folsom (1923). It seems quite probable that some of the cases of tuber cracking and deforming formerly attributed to Rhizoctonia are in reality the effects of some of the degeneration diseases.

Losses from Mosaic.—The losses from mosaic are due to the reduced yields and lowered quality of the stock from diseased hills. As early as 1917, Murphy recognized the heavy losses from mosaic. In a test reported by him, mosaic hills gave 52 to 63 per cent of the yield of normal hills. On account of the small size of the tubers from mosaic hills, only 82.7 per cent were marketable, as contrasted with 91.6 per cent of the tubers from healthy hills. From the case studied, the conclusion was

reached "that for an average crop of 250 bushels per acre, the yield of marketable tubers is reduced about 1⅕ bushels for every 1 per cent of mosaic present." A recent test of the effect of mosaic on the reduction in yield is reported by Folsom and Schultz (1924) and showed that

In pounds per hill, in comparison with the healthy hills, the yield rate was reduced 15 per cent by the presence of about 45 per cent mosaic, 35 per cent by mosaic appearing for the first season in the progeny of healthy plants and 40 per cent by mosaic of 2 or more years standing.

These figures were based on Green Mountain stock, in which most of the mosaic was of the mild type. In rugose mosaic or its extreme type mosaic dwarf, the reduced yields will be more pronounced. Krantz and Bisby (1921) report the rapid decline of yields from a number of varieties, including Gold Coin, Peerless Junior, Early Ohio, Minnesota No. 2 and Burbank, when grown at the University farm. The yields at the beginning of the tests in 1914 varied from 167 to 226 bushels per acre, while in 1919 the same stock had become so diseased that the yields varied from no potatoes to 13 to 29 bushels. Where the same stock was grown at some more favorable location for a part of the time and then brought back to the university farm, the decline was not so rapid. Folsom *et al.* (1926) report 10 per cent reduction from mild mosaic and 50 per cent from rugose mosaic. Gardner and Kendrick (1928) record a decrease in yield of 51 to 82 per cent by the use of infected Cobbler or Bliss seed. These figures will suffice to emphasize that under conditions favorable to the development of mosaic, the disease may be responsible for the complete "running out" of a variety in a few years' time. This experience has been repeated from Maine to the Pacific Northwest.

Etiology.—Mosaic in its various forms is an infectious or communicable trouble of the virus type and is hereditary or perpetuating. When Orton first published on mosaic in 1914, it was thought to be transmitted through the tubers, but definite proof of this was not furnished until later. While the disease was recognized at that time as having certain resemblances to the infectious tobacco mosaic, its infectious character had not been demonstrated. The different kinds of mosaic differ somewhat as to their degrees of infectiousness, but they can all be transmitted from diseased to healthy plants. This transmission may be demonstrated by artificial means and takes place under natural growing conditions in the field. Transmission is successful by (1) stem grafts; (2) tuber grafts (see Leaf Roll for method); (3) juice inoculations; and (4) the feeding punctures of aphids which have been pastured on diseased plants. Potato mosaic is not so infectious as tobacco mosaic, which is transmitted by contact. Mere contact of aerial or underground parts is not sufficient for transmission of potato mosaic, but organic union appears to be necessary. This is indicated by complete failure of transmission when

attempted tuber grafts fail to unite. From this behavior, it will be seen that there is no danger of transmission from tuber to tuber during the storage period, unless sprouting should take place and aphids should be present.

Juice inoculations have been made in a number of different ways, using both filtered and unfiltered juice: (1) by filling a cavity in a seed piece with the juice from crushed stems and leaves of diseased plants; (2) by painting the juice on rubbed, bruised or slashed leaves; (3) by hypodermic injection into stems or petioles; (4) by application of the juice from diseased plants to young healthy leaves, followed by rubbing and crushing of parts of the leaves between the fingers; and (5) by placing a diseased leaf in contact with a healthy one and rubbing and crushing together (Schultz *et al.*, 1919). Varying success followed the use of these methods, some being negative, but (4) and (5) were especially successful. These so-called leaf-mutilation inoculations have been very generally used in transmission studies (Schultz and Folsom, 1923).

Aphid transmission was demonstrated by Schultz *et al.* (1919) by observation of natural dispersal and also by the artificial transfer of the insect vectors, and similar results were later obtained by Quanjer (1920). The most positive proof of insect transmission was obtained by the use of insect-proof cages. In a test made in the winter of 1918–1919 under greenhouse conditions, spinach aphids which had been pastured on mosaic plants were placed on mosaic-free plants under the cages, and, of 18 plants used, 13 developed typical mosaic symptoms, while aphids transferred from healthy potatoes and from radish plants induced no mosaic symptoms.

Symptoms of mosaic were first seen in 18 to 31 days and then consisted of the mottling characteristic of slight mosaic, but the mottling soon became more pronounced and sometimes was accompanied by considerable wrinkling (Schultz *et al.*, 1919).

Quanjer (1920) reports that in his experiments "every plant on which mosaic aphids were fed got mosaic progeny" but that the symptoms of primary disease did not always show during the current season. The testing of the progeny was the certain means of determining whether transmission had taken place.

The following aphids have been demonstrated to be vectors of potato mosaics: *Myzus persicæ*, *M. pseudosolani*, *Macrosiphum gei* (*-solanifolii*) *Aphis rhamni* and *A. fabæ* (*rumicis*) (Schultz and Folsom, 1923; Elze, 1927; Smith, 1927, 1929). Elze reports transfer also by cabbage caterpillars and by the flea beetle (*Psyllioides affinis*). Negative, or uncertain results were obtained with capsids, leaf hoppers, and white flies (Smith, 1927).

Rugose mosaic is the most infectious of the mosaic types, and current-season symptoms in the Cobbler, Rural and Rose groups may be readily

obtained by leaf-mutilation inoculations in either the greenhouse or the open field. Leaf-rolling mosaic is similar to mild mosaic in infectiousness, but both are more easily transmitted than leaf roll.

A frequently observed and important feature of mosaic is the masking of the symptoms as a result of exposure to certain environmental conditions. This relation of temperature to the visible expression of mosaic symptoms was first shown under controlled conditions by Johnson for tobacco mosaic and later for potato mosaic (1922). Plants were grown in the greenhouse in air-control chambers held at fairly constant temperatures for 1 to 3 weeks. The effect on mosaic was based either on the disappearance of mosaic symptoms or on the intensity of their expression.

> Temperatures as low as 6°C. seemingly did not inhibit the disease. Taking the growth of the host into consideration, the optimum temperature lies between 14 and 18°C. Above 20°C., symptoms disappear, the rate or recovery from the disease being increased in proportion to increase of temperature within the limits of host development. To inhibit the disease completely, however, within a period of 1 to 2 weeks, a temperature of 24 to 25°C. is necessary, and this may be regarded as the maximum temperature for mosaic manifestation in the potato. New leaves, free from symptoms, appear quickly at this temperature, and older leaves gradually lose their symptoms, the rate of "recovery" being roughly proportional to the age of the leaf, *i.e.*, the older the leaf the longer the time required for recovery (Johnson, 1922).

In these first studies, the type of mosaic was not indicated, but Goss (1924) has recently noted essentially similar relations for mild mosaic, medium-plus mosaic (rugose) and curly dwarf and has emphasized the fact that masking of symptoms is also favored by low moisture and increased sunlight. The important bearing of these air environmental factors is stressed, since all three—high temperature, low air moisture and intense sunshine—usually occur together under field conditions and in certain environments may be sufficient to cause almost complete masking of symptoms. In later studies on the relation of environment, Goss and Peltier (1925) report that

> The effect of air temperatures on the foliage symptoms of mosaic has again been found to be very pronounced and appears to be the most important factor in the masking of foliage symptoms of the degeneration diseases.
>
> Mottling was again found to be the most constant symptom of mosaic at all temperatures, although a change in the type and degree of mottling was clearly evident. With mild mosaic mottling did not appear on new growth at 25°C., while the distinct mottling occurring at 15°C. became very indistinct or diffuse when the plants were changed to 25°. The indistinct or diffuse mottling of plants affected by rugose mosaic was not greatly changed at high temperatures.
>
> The wrinkling, ruffling, rugosity, curling, rolling and brittleness of mosaic plants all tended to disappear at 25°C. In addition to the masking of the above symptoms which has previously been recorded, it was found that the streaking,

spotting, burning and leaf dropping of rugose mosaic did not occur at 25°C. on Bliss Triumphs.

The symptoms of mild mosaic are so greatly masked at high temperatures that the plant often looks healthy, while the other mosaic types and combinations of two mosaics or mosaic and spindle tuber still retain enough of the symptoms at 25°C. to distinguish them clearly from mild mosaic and healthy plants. The identification of the individual diseases, however, is very difficult and often impossible at 25°C.

In a later study on the relation of environmental factors to potato mosaic, Tompkins (1926) discussed the effect of air temperature, soil temperature, soil moisture, air humidity, light and nutrition and reported that air temperature was the main factor affecting the expression of mosaic symptoms. Short exposures to temperatures above the critical (23 to 24°C.) were sufficient to mask mosaic symptoms, the rate of masking depending on the duration of the high temperature, the actual temperature prevailing and the age of the leaves.

The exposure of mosaic plants to the high temperatures does not appear to destroy the infectious properties of the mosaic virus, since aphid transmission may continue during the period of masking, and progeny from all such diseased hills will again exhibit the disease when grown under favorable temperature conditions.

Varietal Relations.—With some modifications of symptoms, the various types of mosaic appear to be generally capable of intervarietal transmission or of transmission from recognized varieties to seedlings or from seedlings back to the same varieties or to others. Quanjer (1922) reports that mosaic (apparently mild mosaic) can be transmitted to other solanaceous species, *e.g.*, tomato and tobacco, by grafting. Schultz and Folsom have obtained similar results by leaf mutilation and by aphid transmission but conclude that potato mild mosaic is not identical with tobacco mosaic and that the tomato is suceptible to both of these mosaics and also to potato rugose mosaic. Mild mosaic has also been transmitted to black nightshade (*Solanum nigrum*). More recently, Elmer (1925) has reported the successful transfer of mosaic from potatoes to cowpeas by means of aphids, but artificial inoculations were unsuccessful, which would lead one to question the reliability of the data on aphid transfer.

Not very much can be said at the present with reference to the resistance of varieties. While variations have been noted in suceptibility, no immune strains of varieties have been found. Seedlings appear to be quite generally susceptible. A large amount of the experimental work has been conducted with the very susceptible Green Mountains and Bliss Triumphs. It is stated that the Irish Cobbler is practically free from mosaic in northern Maine (Folsom 1920) and that other varieties resemble the Cobblers. Murphy (1922) mentions eight varieties, including Great Scot, as resistant to mosaic in Ireland, Great Scot being the only one that

withstands leaf roll also. It is possible that high resistance to mosaics may be developed by selection among known varieties or in seedling strains, but the outlook is not very promising.

Control.—The control of mosaic is more difficult than that of leaf roll, because of its more ready transmission and the pronounced masking of symptoms. The recommendations for leaf-roll control are of general application in holding the losses from mosaic down to a minimum. Two lines of procedure are open to the potato grower: (1) to rely on seed produced in regions in which mosaic is of little consequence or where extreme care is taken by professional seed growers to produce high-grade stock; or (2) to produce his own seed in an isolated seed plot, as outlined under Leaf Roll. Experience has shown that even greater care must be practiced in rogueing the seed plot, beginning early before the mosaic symptoms are masked. The success attending the seed-plot method will vary in different environments, and experience must be the guide as to its value. An experiment reported by Stewart (1924) is illustrative of the degree of success which may attend the seed-plot method and rogueing. Greenhouse-tested, mosaic-free Green Mountain seed was grown in 1921, 1922 and 1923 in isolated plots and carefully rogued, but the 1923 crop when tested in the greenhouse showed 4.4 per cent of mosaic. In case the use of imported certified seed is adopted as the best practice, it may be possible to use stock from this crop the second season and use the certified seed only every other year.

In every country, a search should be made for secluded or isolated localities in which seed-potato production can be profitably established because of freedom from the insect vectors. Cases are on record where the same seed stock has been grown for 25 years without showing any signs of deterioration. It is now generally conceded that "running out," or degeneration, of potatoes is not a physiological process but the result of the numerous virus diseases and that seed stock may be kept up indefinitely if these diseases are absent or can be excluded.

The recommendation that all wild rose bushes which might harbor aphids should be removed may be practical in some environments, but in many regions it would be but an idle dream except as applied to the isolated seed plot.

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POTATO LEAF ROLL

This is one of the several degeneration or virus diseases of the potato in which a pronounced rolling of the leaves is a characteristic symptom. Affected plants are not killed, but marked reductions in yield result. The disease is known in Germany as the "Blattrollkrankheit," in France as "la maladie de l'enroulement" and in America and other English-speaking countries as leaf roll, although "curl" and "leaf curl" have been used to a limited extent.

History and Geographic Distribution.—It seems probable that leaf roll and other virus diseases of the potato were prevalent many years ago (see potato mosaic). The opinion has been expressed that the notable failures of potatoes in middle and western Europe from 1770–1780 were due to leaf roll and diseases of the mosaic group. Leaf roll as a specific disease of the potato, however, was first recognized in Germany and Denmark in 1905. Previous to this time, the disease had been included with potato troubles known under the general name of "Kräuselkrankheiten." In 1907, a more general outbreak occurred in Germany and from that time on scientific workers have given much attention to this and related diseases. In 1911, there was an outbreak of leaf roll in northern Colorado and in western Nebraska, and the first clear-cut presentation of the leaf roll as a potato disease in America was published by Orton (1914). This was made possible through a study of potato diseases in Europe in 1911 and extensive travel later through the important potato sections of America. Even as early as 1914, Orton wrote: "The literature on leaf roll has become so voluminous that few will undertake to peruse all the contributions, which are, indeed, of very uneven merit, and

anyone who attempts it is likely to emerge with his concepts of the disease more confused and hazy than at the start." This bewildering condition was due to the imperfect recognition of the symptoms of the disease and to the fact that there were so many and varied opinions as to its true cause.

There are a number of important steps in the history of the disease: (1) the recognition of leaf roll as a specific disease (1905); (2) the establishment of its heritable character; (3) the histological studies showing phloëm necrosis as a constant internal character (Quanjer, 1913; Artschwager, 1918); (4) the demonstration of its communicability by grafting (Quanjer *et al.*, 1916; Schultz and Folsom, 1919); (5) the proof of transmission by aphids (Botjes, 1920; Schultz and Folsom, 1921); (6) the observation of net necrosis and spindling sprout as a frequent accompaniment (Schultz and Folsom, 1921); and (7) the proof that insect transmission may not be evident until the second season and that the disease transmitted to other plants by grafting (tomato, tobacco, etc.) may remain latent—in other words, that these may be infection carriers without themselves showing the disease (Quanjer, 1923). During recent years, workers in Great Britain, continental Europe and America have made numerous contributions to our knowledge of leaf roll, and it has also been studied by Japanese investigators (Kasai, 1921).

Exact data as to the detailed geographic distribution of leaf roll are not available, but from the numerous reports it seems probable that its occurrence is coexistent with the cultivation of potatoes but that it reaches its greatest severity only in regions in which its insect carriers are especially abundant. These carriers appear to decrease in the northern latitudes or higher altitudes, which would explain the greater freedom of cooler sections from leaf roll. This is offered as the explanation for more infrequent occurrence of the disease in much of north Scotland than in the south of England, and similar relations may be pointed out for the American occurrence of leaf roll. Since no visible organism is connected with the disease, and since other non-parasitic conditions and several parasitic invasions may be responsible for somewhat similar symptoms, the presence of leaf roll has been frequently overlooked, unless special studies have been made by workers who have become thoroughly familiar with the disease.

Symptoms and Effects.—The most common symptom is the characteristic *rolling of the leaves*, which has suggested the common name of the disease. The leaflets curl upward from the margin toward the midrib and in the extreme cases becomes nearly tubular. The *texture* of the rolled leaves is different from that of normal leaves.

The rigid character of the affected leaf or leaflet is a *very important diagnostic character*. The "feel" is hard and crisp. The terms "brittle," "turgescent," "rigid," "leathery" have been used to describe this condition. Leaves showing symptoms of leaf roll do not wilt and become limp during drought, as normal leaves do (Wortley, 1918).

The petioles of affected leaves are frequently raised to form a more acute angle with the stalk than in normal plants, especially in the case of primary leaf roll. When infected tubers are planted, the lower leaves invariably show the first rolling of the leaflets, and the trouble advances until the entire plant may show the symptom. Where the disease is contracted by a plant during the growing season, the rolling of the leaflets may be confined to the upper parts of the plant (primary leaf roll); and when this symptom is only slightly expressed, the definite diagnosis of leaf

roll is very difficult and sometimes cannot be determined with certainty until the progeny has been grown the next season.

At this point, it will be well to note that a rolling of the leaflets may be caused by other agencies than the true leaf-roll disease: (1) water-logged soil; (2) drought or extreme heat; (3) excessive quantities of fertilizer, especially potash; (4) the bacterial disease blackleg; (5) fungous diseases like Fusarium or Verticillium wilts and Rhizoctonia; and (6) several other virus diseases of the potato (see list under Mosaic). Careful attention to the detailed symptoms in the various cases must frequently be given to make a certain diagnosis. Too much weight should not be attached to the presence of a parasite like Rhizoctonia, for example, since this fungus is frequently in evidence on plants affected with the leaf roll.

FIG. 82.—Potato plant showing a well-developed case of leaf roll. (*Photograph by B. F. Dana.*)

The *color of the foliage* deviates more or less from that of normal plants but varies with the variety, the environmental conditions and the severity of the infection. Early stages of leaf roll may show but a slight pallor of the foliage, while as the season advances, especially under conditions of extreme drought, the color may be a pronounced yellowish. In many cases, the affected leaves will show reddish or purplish colors, which are conspicuous in certain varieties or in leaf-roll infections of long standing.

The *type of growth* of the plant is modified, but the change is not a constant character. In some cases, the affected plant is more slender, with an abnormally erect V-shaped form, while in others a low-spreading, truncate or compressed form may be assumed. In leaf roll from infected

tubers, the length of life of the diseased plants appears to be shortened, and they are generally smaller than normal.

The *endurance of the seed piece* has frequently been mentioned as a character of leaf roll, but this does not seem to be a character of diagnostic value, since it has been pointed out that under certain conditions the seed pieces of normal plants may persist; while under conditions very favorable

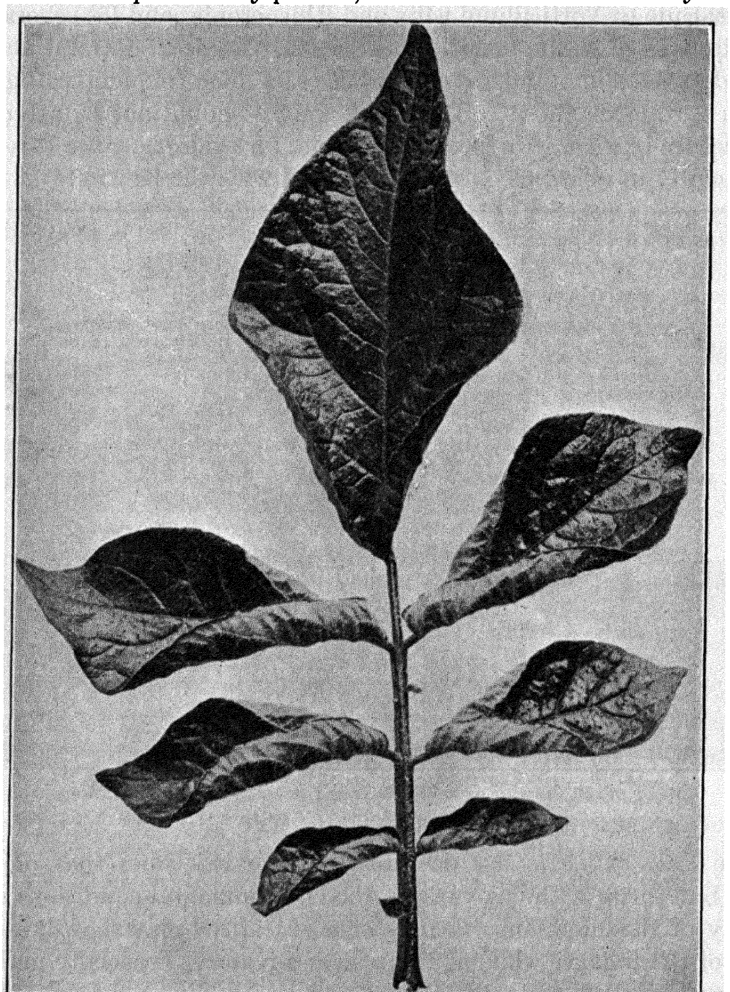

FIG. 83.—Single potato leaf showing the characteristic rolling of the leaflets from an affected plant. (*Photograph by B. F. Dana.*)

for decay, leaf-roll seed pieces may disintegrate. Sound seed pieces are also found in some of the other virus diseases.

The *reduction in the size of the tubers* is an invariable feature in the disease, and very frequently the *stolons are short*, so that the tubers are borne in clusters close to the stem or directly upon it. There may be a

few moderate-sized tubers or the tubers may be fairly numerous and small.

The *necrosis of the phloëm* has been especially emphasized as a symptom of leaf roll by Quanjer (1913, 1920), who suggests that the disease "may better be termed phloëm necrosis or, according to Pethybridge, leptonecrosis." The findings of Quanjer were corroborated by Artschwager (1918), and in later studies (1923) he states that

> Stem sections of a typical leaf-roll plant exhibit, as a diagnostic internal symptom, a necrosis and lignification of the phloëm groups. In the case of severe external symptoms, the diseased groups pervade the entire plant, with the occasional exception of the underground organs. The distal stem region is commonly affected, and in nearly every instance the necrotic changes are of an extreme type. The basal stem region always shows necrotic changes when external symptoms become evident while the plant is still young. As a rule, necrosis of the phloëm in the lower stem means generally necrosis of the plant throughout its extent, but the symptoms may decrease toward the distal end or disappear altogether. At any given height of the stem, the node is typically more severely affected than is the internode. This condition is especially observed in the initial stages of the disease, but during subsequent development either region may be equally affected . . . While obliteration of the phloëm is always observed in connection with leaf roll, it is also an accompanying phenomenon in other diseases. It is not so much its mere presence as its universality in distribution, coupled with the absence of necrosis in other tissues, which gives it a real diagnostic value.

Net necrosis of tubers and spindling sprout have recently been recorded as symptoms accompanying leaf roll (Schultz and Folsom, 1921).

> Net necrosis is apparently a leaf-roll symptom, being a discoloration which results from tuber phloëm necrosis and which appears more often as conditions of variety, recency of infection and weight of tuber are more favorable. It develops in the dormant tubers without relation to differences in the storage temperature. When it occurs as a symptom of leaf roll, the effects of the latter are still more detrimental, one being a decided spindliness of the sprouts.

Later studies by Gilbert (1928) and Elze and Quanjer (1929) have shown positively that net necrosis of tubers is a first-season symptom following infection with leaf roll in American varieties. German varieties like Roode Star or Duke of York exposed to the same source of infection showed phloëm necrosis confined to stalks and leaves (Elze and Quanjer, 1929). The relation of spindling sprout to leaf roll has been denied by Atanasoff (1926) and Elze (1927). The latter reports successful transmission of spindling sprout with *Myzus persicæ* but is inclined to view this symptom as the result of a distinct virus. The net necrosis discussed by Atanasoff (1926) as connected with aucuba mosaic is apparently the pseudo-net necrosis of Quanjer and is caused by another virus entity.

Neither net necrosis nor spindling sprout can be accepted as diagnostic characters for leaf roll, since they are not always in evidence and

may be caused by entirely different factors (unfavorable temperatures, etc.).

The *accumulation of starch in the leaves* of leaf-roll plants can be demonstrated by the standard iodine test. In a normal plant, translocation of carbohydrates proceeds during the night period, and in the morning the leaves are devoid of starch; while in leaf-roll plants, the starch is not carried to tubers, or only slowly, and the leaves are still full of starch.

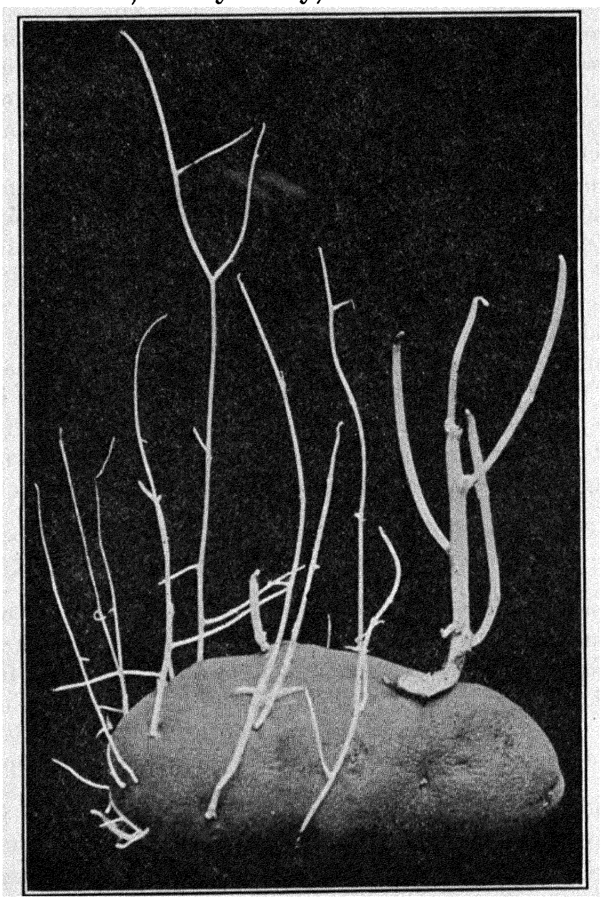

FIG. 84.—Spindling sprout, a symptom which frequently accompanies leaf roll. (*Photograph by B. F. Dana.*)

In recently infected plants, there is a little phloëm necrosis in the upper part of the stalk and the upper leaves. In such cases, the translocation of the starch is inhibited in the upper structures, while in the basal portions normal conditions prevail. It has been concluded that the starch accumulation is caused by a disturbed transport and not by a modification of enzymatic processes (Thung, 1928).

Quanjer (1923) has recognized a second form of leaf roll which he has designated "marginal leaf roll." In this disease,

The margins only of the leaflets are shortly curled upwards, often more in the upper part than in the lower portion of the plant. Leaflets do not take the upright habit. Transport of starch only prohibited in margins. No phloëm necrosis in midribs, petioles and stems.

It has been suggested that this is identical with spindle tuber, which shows marginal rolling of the upper leaflets in some localities.

A third form of leaf roll has been recognized by Schultz and Bonde (1929). "Infected plants slightly dwarfed manifest roll of the upper leaves similar to primary leaf roll . . . However, apical leaf roll is distinguished from primary leaf roll by persisting only on the upper leaves in succeeding generations."

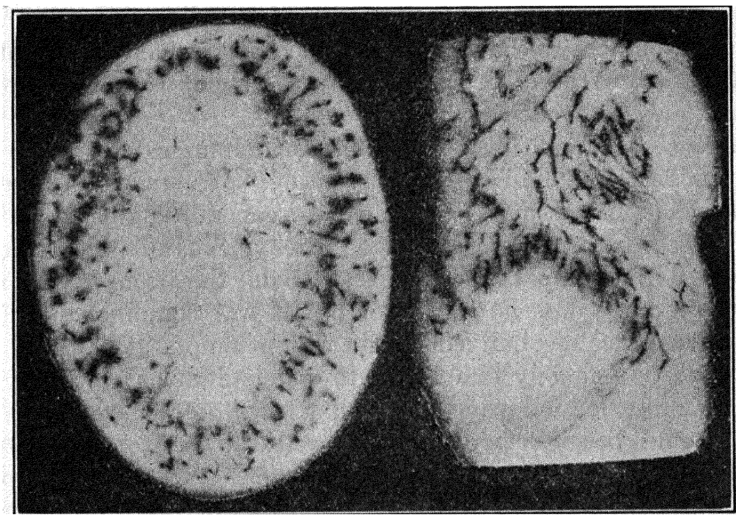

FIG. 85.—Cross and longitudinal sections of potato tubers showing net type of phloëm necrosis.

Losses from Leaf Roll.—Leaf roll is but one of the virus diseases that may be responsible for the "running out" of potato seed stock. When fields become infected with the disease, it is only a question of time when production will sink to a very low point. It has been a common thing to say that leaf-roll stock will reduce the yield 50 per cent or more. Under favorable conditions for the spread of leaf roll, stock only slightly infected may become worthless in 2 to 4 years. The tabulation on page 308 of reported yield reductions is taken from the report of Gardner and Kendrick (1924).

Whitehead and Currie (1931) report reduced weights of tubers as a result of leaf-roll infection from a minimum of 14 per cent in Up-to-date to a maximum reduction of 97.6 per cent in Herald.

The reduction in total yield is very high, while still further losses are inflicted because the stock is not all marketable. These figures are

Authority	Place	Per cent of yield reduction
Murphy	Canada	66–82
Schultz and Folsom	Maine	42
Hungerford	Idaho	80
Cotton	Great Britain	36–75
Whitehead	Ireland	45–79
Murphy	Ireland	59–79
Gram	Denmark	20–89
Gardner and Kendrick	Indiana	22–66

sufficient to emphasize the extremely heavy losses which may result from leaf roll alone. When other virus diseases or fungous troubles are present, the losses may be even greater.

Etiology.—Leaf roll is an infectious or communicable trouble of the virus type and is hereditary or perpetuating. When Orton summarized our knowledge of the disease, it had been generally agreed that tubers from diseased plants produce diseased progeny, a character distinguishing true leaf roll from temporary or false leaf roll due to various environmental factors, but there was no evidence that it was communicable (1914). The negative results obtained in the first attempts at artificial transmission were due mainly to a failure to understand the necessary length of incubation period following an inoculation before the symptoms of the disease would become evident. Leaf roll can be communicated by grafting, and two different methods have been successfully employed: (1) stalk grafting and (2) tuber grafting. In tests made in the summer of 1919 (Schultz and Folsom, 1921), stalks from diseased plants were grafted on healthy stalks, using both Green Mountain and Irish Cobbler; and by the end of the growing season, shoots from the stock just below the graft were showing typical leaf roll. Tuber grafts were made by bringing into contact the freshly cut surfaces of halves of healthy and leaf-roll tubers and holding them tightly so that organic union could take place. At the end of the growing season, leaf roll had been transmitted to shoots from the healthy halves in all cases in which there was an organic union of the pieces. Recent work (Elze, 1931) indicates the possibility of transmission through seed from infected mother plants.

An important forward step in our knowledge of the etiology of leaf roll was made by Botjes in Holland (1920), when he demonstrated that the disease can be transmitted from diseased to healthy plants by the feeding punctures of aphids. This method of transmission was later confirmed by Schultz and Folsom (1921). Using the same method that had proved successful in the transmission of mosaic, they

took aphids from leaf-roll plants and kept them on healthy plants under insect-proof cages. The controls remained healthy, but the plants on which the aphids were pastured developed the first symptoms of leaf roll after 24 to 29 days, while typical symptoms of severe leaf roll followed with the progress of the infections. Field observations have served to confirm the importance of aphid transmission as a natural method of dissemination. Numerous observations have shown that the field spread of leaf roll is much more rapid in some localities than in others (Gardner and Kendrick, 1924); the reason for this was not understood for some time, but at the present the abundance of the vectors is recognized as the cause. Recent work (Smith, 1929) has shown that the incidence of infection is much greater with colonies of 12 to 18 aphids per plant than with only 2 to 6. Transmission tests (Elze, 1927, 1931; Smith, 1929; Murphy and McKay, 1929; Whitehead, 1931) point to *Myzus persicæ* as the most efficient vector of leaf roll. Other aphids are also capable of carrying leaf roll (*M. circumflexus, Aphis rhamni, A. fabæ* and possibly others) but may be considered as poor vectors. Elze (1931) suggests that the efficiency of *M. persicæ* is due to definite biological relation with the virus as indicated by (1) the existence of an incubation period for the virus in the insect; (2) the retention of infective power after moulting; (3) the retention of infective power after feeding for a long period on plants not susceptible to leaf roll; and (4) the certainty of infection from a small number of viruliferous individuals. Smith (1929) thinks that *M. persicæ* remains viruliferous throughout life. The rather infrequent transmission by biting insects would appear to be mainly of mechanical nature.

Quanjer (1920) believed that he had obtained evidence that there is a soil contamination in leaf roll and that infection of a crop may result from the infective principle that persists in the soil. Later tests by others have led to the belief that these apparently positive results were due to ungathered diseased tubers which lived over winter and produced infected plants from which aphids carried the disease to the healthy stock. Leaf roll is transmitted with more difficulty than any of the other virus diseases of the potato. It is generally agreed that leaf roll cannot be communicated by mere contact of either roots or tops, yet Whitehead (1923) has presented evidence of communication from healthy to normal plants through the soil. All attempts to transmit it by leaf mutilation or by juice inoculation, methods which have proved so successful in mosaic transmission, have given only negative results.

Previous to the definite recognition of leaf roll as due to the transmission of a virus or infective principle, many different theories were advanced to explain the disease. Some of these were as follows: (1) either excesses or deficiencies of mineral elements in natural soils or the use of fertilizers; (2) the use of either unripe or matured tubers for

seed or of tubers from prematurely ripened plants; (3) poor cultural methods; (4) varietal degeneration due to continued vegetative propagation with failure to practice seed selection; (5) the invasion of a parasitic fungus, probably a Fusarium. The presence of fungi on the roots or stems of leaf-roll plants has been more or less confusing, since these were frequently reputed parasites, but numerous cases of fungus-free plants affected with leaf roll pointed to the fungi as either secondary invaders or pathogenes working simultaneously with the causal agent of leaf roll. Even in recent time, some workers have refused to accept the view that leaf roll is caused by a virus (Schweizer, 1930; Merkenschlager, 1930).

One of the important features in leaf roll which has been brought out by the later investigators is the fact that transmission by aphids may not become evident during the current growing season but that the infective principle is carried down into the tubers, so that the disease will appear the next season if these tubers are planted. According to recent work (Whitehead and Currie, 1930), infections of Aaran Comrade 8 or less weeks after planting will result in secondary symptoms, while later infections will give only primary symptoms, and for Kerr's Pink the infection must be within 5 weeks after planting to yield secondary symptoms. In this connection, mention should be made of the infection of sprouting tubers by aphis vectors (Stewart and Glasgow, 1930). If the results reported by Quanjer, that the disease may be transmitted from leaf-roll plants to tomato and other solanaceous species and back to the potato by grafting, without the symptoms of the disease appearing in these solanaceous forms, should prove true in the case of natural insect transmission, control of leaf roll would be even more complicated.

In primary infections, not all of the tubers produced by a plant carry the disease. It has even been shown that some of the tubers from the same stalk may be healthy while others are infected and, more rarely, that sprouts from certain eyes of a tuber may produce leaf-roll plants while other sprouts from the same tuber remain healthy. It seems probable that this variable behavior is due, in part at least, to the length of time that has elapsed between inoculation and the death of the plants or the harvesting of the tubers, since it has been shown that the virus passes down through the plant rather slowly.

The distance which leaf roll will spread in the field is rather limited. The greatest spread will be to plants immediately adjacent to infected ones, but it has been reported to spread across at least three to four rows. The percentage of infection in rows adjacent to a row showing 100 per cent leaf roll have been noted as follows: first row, 87; second row, 9; third row 3 (Murphy and McKay, 1927). Some field observations have indicated spread for greater distances, up to 75 to 150 feet, but at such distance the infection is low. From all the observations, the conclusion

may be reached that the danger of spread from one field to another is very slight, if the fields are well separated.

Variety Resistance.—Differences in varietal susceptibility have been reported by various workers, but as yet no immune varieties have been found. Some of the cases of apparent resistance are probably due to early maturity, which removes many of the opportunities for infection, since aphids have not reached maximum development at the time the crop is ready for harvest. This is the apparent explanation for the greater freedom of Early Ohio and Irish Cobbler as contrasted with the late Rural varieties. A number of investigators have reported Early Ohios, Cobblers and Rurals as very susceptible, but some field tests have shown a greater seasonal spread to Rural New Yorker when all varieties were equally exposed to infection (Gardner and Kendrick, 1924). Cotton (1922) lists many commercial English varieties as susceptible but states that Great Scot is very seldom infected. The great variation in the yield of different varieties equally exposed to infection (14 to 97.6 per cent) offers hope for the production of high-yielding and resistant strains by selection and breeding (Whitehead and Currie, 1931).

Control.—The problem of leaf-roll control is inseparably connected with the control of mosaic and various other virus diseases, since leaf roll is rarely the only virus disease present in any environment. The procedure to follow will be somewhat different for growers of table stock and producers of seed. In a region in which leaf roll is known to spread readily, it is not advisable for a grower to attempt to produce his own seed. The use of an isolated seed plot, planted with selected seed of good size, free from internal necrosis, may hold up the stock for a time, if the plot is carefully rogued, sprayed at intervals for the control of aphids and harvested early, but in the majority of cases the grower of table stock will find it more profitable to rely on the use of seed from sections in which leaf roll is less prevalent, and the greatest safety should attend the use of certified seed from such regions. The value of seed stock from the potato-seed sections of Michigan, Wisconsin and Minnesota does not depend so much on seed certification as upon the lesser prevalence of leaf roll and other virus diseases.

Some of the features which should be especially emphasized in obtaining and maintaining seed stock are as follows: (1) Field selection of high-yielding hills will not eliminate leaf roll if the disease is present in the plot, since primary infections which are not discernible may have taken place; (2) selected seed for starting a seed plot may be tuber indexed by growing one eye from each tuber in the greenhouse (also in the field, 6 to 8 weeks being required), and the presence or absence of leaf roll determined; (3) isolation of the seed plot is necessary to prevent the introduction of leaf roll by insect carriers; (4) the destruction of volunteer potatoes is of importance, since these may be already infected and furnish the virus to

be carried to the seed stock by aphids; (5) rogueing for leaf roll is more effective than for mosaic, since its spread is slower and masking of symptoms less frequent; (6) primary- or current-season infections do not reduce the yield but render the crop of little value for seed purposes; (7) the spread of leaf roll is directly proportional to the abundance of aphids, and their control should receive attention by seed growers.

According to Schweizer (1930), promising results in control have been obtained by the application to the soil of a mixture of manganese, lime, cyanide and uranium salts at the rate of 500 cubic centimeters per plant or by soaking the tubers for 5 minutes in the same solution. It would seem doubtful whether the author in this case was working with a true virosis.

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IMPORTANT VIRUS DISEASES[1]

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Abutilon (*Infectious chlorosis*).—(See brief consideration, p. 263.)

Alfalfa (*Dwarf*).—WEIMER, J. L.: Alfalfa dwarf, a hitherto unreported disease. *Phytopath.* **21**: 71–75. 1931. (See Legumes and Tobacco (Ring spot).)

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[1] This list has been compiled by the author and Dr. Grover Burnett, Research Assistant in Plant Pathology.

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Cabbage (*Mosaic*).—(See Crucifer (Mosaic).)

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Corn (*Streak or variegation*).—STOREY, H. H.: Streak disease, an infectious chlorosis of sugar cane, not identical with mosaic. *Imp. Bot. Conf.* **1924**: 132–144. 1925. ——: The transmission of streak disease of maize by the leafhopper *Balclutha mbila*. *Ann. Appl. Biol.* **12**: 422–439. 1925. Also *Ann. App. Biol.* **15**: 1–25. 1928. The only successful method of transmission is by the leaf hopper *B. mbila*.

Corn (*Sugar-cane mosaic*).—BRANDES, E. W.: Mosaic disease of corn. *Jour. Agr. Res.* **19**: 517–521. 1920. STONEBERG, H.: The productiveness of corn as influenced by the mosaic disease. *U. S. Dept. Agr. Tech. Bul.* **10**: 1–18. 1927. *Aphis maydis* is an important vector.

Cosmos (*Yellows*).—(See Aster (Yellows).)

Cotton (*Acromania or crazy top*).—COOK, O. F.: Acromania or "crazy top," a growth disorder of cotton. *Jour. Agr. Res.* **28**: 803–828. 1924. KING, C. J. and LOOMIS, H. F.: Factors influencing the crazy-top disorder of cotton. *U. S. Dept. Agr. Bul.* **1484**: 1–21. 1927. Symptoms of this disease which has not yet been proved to be of a virus character are compared to brachysm, tomosis, hybosis, cyrtosis and stenosis.

Cotton (*Leaf curl*).—JONES, G. H. AND MASON, T. G.: On two obscure diseases of cotton. *Ann. Bot.* **40**: 759–772. 1926. Transmitted by grafting and by an unidentified species of white fly (Aleurodidæ). GOLDING, F. D.: A vector of leaf curl of cotton in southern Nigeria. *Empire Cotton Growing Rev.* **7**: 120–126. 1930.

Cotton (*Stenosis or smalling*).—COOK, O. F.: Malformation of cotton plants in Haiti. *Jour. Hered.* **14**: 323–335. 1923.

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Cranberry (*False blossom*).—DUBROSCKY, I. D.: Studies on cranberry false-blossom disease and its insect vector. *Contr. Boyce Thompson Inst. Plant Res.* **3**: 59–83. 1931. The vector is the blunt-nosed leaf-hopper (*Euscelis straitulus*).

Crocus (*Mosaic*).—(See Hyacinth (Mosaic).)

Crucifers (*Mosaic*).—CLAYTON, E. E.: A study of the mosaic disease of crucifers. *Jour. Agr. Res.* **40**: 263–270. 1930. Affects turnips, rutabagas, Brussels

sprouts, cauliflower, mustard, rape and Chinese cabbage, but cabbage is highly resistant or immune.

Cucumber (*Mosaic*).—Various phases of the disease have suggested the common names of mottled-leaf mosaic, wart disease, nubbin and white pickle. DOOLITTLE, S. P.: The mosaic disease of Cucurbits. *U. S. Dept. Agr. Bul.* **879**: 1–69. 1920. —— AND WALKER, M. N.: Further studies on overwintering and dissemination of cucurbit mosaic. *Jour. Agr. Res.* **31**: 1–58. 1925. WALKER, M. N.: A comparative study of the mosaic diseases of cucumber, tomato and Physalis. *Phytopath* **16**: 431–458. 1926. HOGGAN, I. A.: The peach aphid (*Myzus persicæ* Sulz.) as an agent in virus transmission. *Phytopath.* **19**: 109–123. 1929. PORTER, R. H.: A new mosaic disease of cucumber. *Phytopath.* **20**: 113. 1930. All cucurbits except the Chinese Long cucumber and watermelons are susceptible to ordinary or white pickle mosaic, but a new mosaic, the "Bettendorf," infects the Chinese Long, watermelons and citron, the symptomology being different.

Currant (*Reversion or nettlehead*).—AMOS, J. AND HATTON, R. G.: Reversion of black currants. I. *Jour. Pomol. Hort. Sci.* **6**: 167–183. 1927. —— AND ——: Reversion in black currants. II. *Jour. Pomol. Hort. Sci.* **6**: 282–295. 1928. The vector appears to be the big bud mite (*Eriophyes ribis*).

Daffodil (*Mosaic, yellow stripe, or gray disease*).—GRIFFITHS, D.: *In* Daffodils. *U. S. Dept. Agr. Circ.* **122**: 62–63. 1930.

Dahlia (*Mosaic and dwarf or stunt*).—GOLDSTEIN, BESSIE: The x-bodies in the cells of dahlia plants affected with mosaic disease and dwarf. *Bul. Torr. Bot. Club* **54**: 285–293. 1927. BRANDENBERG, E. V.: Ueber Mosaikkrankheiten an Compositen. *Forsch. Gebiet Pflanzenkr. u. Immunität Pflanzenr.* **5**: 39–72. 1928.

Dodonæa (*Spike disease*).—SASTRI, B. N. AND NARAYANA, N.: The spike-disease of *Dodonæa viscosa*. *Jour. Ind. Inst. Sci.* **13A**: 147–152. 1931.

Egg plant (*Mosaic*).—BURGER, O. F.: Report of the Plant Pathologist. *Fla. Agr. Exp. Sta. Rept.* 1924: 84. 1925.

Euonymus (*Infectious chlorosis*).—(See brief consideration, p. 263.)

Freesia (*Yellows*?).—LONGFORD, H. G.: A new disease of Freesia. *Gard. Chron.* **81**: 118. 1927.

Geranium (*Crinkle mosaic*).—PAPE, H.: *In* Krankheiten und Schädlinge der Zierpflanzen, pp. 270–271. Paul Parey, Berlin. 1932.

Gladiolus (*Mosaic*).—DOSDALL, L.: A mosaic disease of gladiolus. *Phytopath.* **18**: 215–217. 1928.

Grape (*Leaf roll*).—PETRI, L.: Sulle cause della arricciamento della vite. *Boll. R. Staz. Pat. Veg. N. S.* **9**: 101–130. 1929. ——: Sull'arricciamento della vite. *Boll. R. Staz. Pat. Veg. N. S.* **11**: 61–83. 1931. Thought to be identical with roncet, court noué and Reisigkrankheit.

Grape (*Mosaic*).—SMOLÁK, J.: Abs. *Rev. App. Myc.* **6**: 213. 1927.

Grape hyacinth (*Mosaic*).—(See Hyacinth (Mosaic).)

Hippeastrum (*Mosaic*).—HOLMES, F. O.: Cytological study of the intracellular body characteristic of Hippeastrum mosaic. *Bot. Gaz.* **86**: 50–58. 1928.

Hop (*Mosaic*).—SALMON, E. S.: The "mosaic" disease of the hop. *Jour. Min. Agr. Gt. Brit.* **29**: 927–934. 1923. BLATTNÝ, C.: Abst. in *Rev. App. Myc.* **6**: 692. 1927. This author lists the following types: aucuba, "squirt" mosaic, mottled mosaic, hereditary sterility, yellow-spot mosaic and hop curl (the last possibly identical with nettlehead). SALMON, E. S. AND WARE, W. M.: The mosaic disease of the hop. I. *Ann. Appl. Biol.* **15**: 342–351. 1928. MACKENZIE, D. *et al.*: ——. II. *Ann. Appl. Biol.* **16**: 359–381. 1929.

Hop (*Nettlehead*).—Two forms are distinguished—*true* and *false*. DUFFIELD, C. A. W.: Nettlehead in hops. *Ann. Appl. Biol.* **12**: 536–543. 1925. A disease thought to be identical with "false nettlehead" has been described in Tasmania under the name of "take-all." *Aust. Jour. Exp. Biol. Med. Sci.* **8**: 9–44. 1931.

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Hyacinth (*Mosaic*).—ATANASOFF, D.: Mosaic disease of flower-bulb plants. *Bul. Soc. Bot. Bulgaria* **2**: 51–60. 1928.

Iris (*Mosaic*).—MARTIN, G. H.: *Plant Disease Rep. Supp.* **73**: 387. 1929. See also ATANASOFF, D.: *Loc. cit.*

Jackbean (*Mosaic*).—UPPAL, B. N.: A new virus disease of *Dolichos biflorus. Intern. Bul. Plant Prot.* **5**: 163. 1931.

Legumes (*Mosaic*).—General for the family. MERKEL, L.: Beitrag zur Kenntnis der Mosaikkrankheit der Papilionaceæ. *Zeitschr. Pflanzenkr.* **39**: 289–347. 1929.

Lettuce (*Mosaic*).—JAGGER, I. C.: A transmissible mosaic disease of lettuce. *Jour. Agr. Res.* **20**: 737–739. 1921.

Lettuce (*Yellows*).—Known in New York as white heart or rabbit ear and in Texas as the Rio Grande disease. Caused by the virus of aster yellows. SEVERIN, H. H. P.: Yellows disease of celery, lettuce and other plants, transmitted by *Cicadula sexnotata. Hilgardia* **3**: 543–571. 1929.

Lily (*Mosaic*).—GUTERMAN, C. E. F.: Diseases of lilies. *Yearbook Hort. Soc. N. Y.* **1930**: 51–102.

Lily (*Yellow flat or rosette*).—OGILVIE, L.: A transmissible virus disease of the Easter lily. *Ann. Appl. Biol.* **15**: 540–562. 1928. GUTERMAN, C. E. F.: *Loc. cit.* Confined mainly to *L. longiflorum* and its varieties.

Lily of the valley (*Mosaic*).—BLATTNY, C.: Mosaika Konvalinky (*Convallaria majales* L.) *Ochrana Rostlin* **9**: 19–21. 1929.

Loganberry (*Dwarf*).—(See Blackberry.)

Marigold (*Yellows*).—(See Aster (Yellows).)

Melon (*Mosaic*).—DOOLITTLE, S. P.: The mosaic disease of melons and cucumber. *Iowa State Hort. Soc. Rept.* **57**: 393–396. 1923. (See also Cucumber.)

Mustard (*Mosaic*).—(See Crucifer (Mosaic).)

Narcissus (*Mosaic*).—(See Hyacinth (Mosaic).)

Nicotiana (*Mosaic*).—WALKER, M. N.: Studies on the mosaic disease of *Nicotiana glutinosa. Phytopath.* **15**: 543–547. 1925. Not distinct from tobacco mosaic as previously reported.

Ocean spray (*Witches' broom*).—ZELLER, S. M.: A witches' broom of ocean spray (*Holodiscus discolor*). *Phytopath.* **21**: 923–925. 1931.

Okra (*Mosaic*).—KULKARNI, G. S.: Mosaic and other related diseases in the Bombay Presidency. *Poona Agr. Coll. Mag.* **16**: 6–12. 1924.

Onion (*Yellow dwarf*).—MELHUS, I. E. *et al.*: A new virus disease epidemic on onions. *Phytopath.* **19**: 73–77. 1929.

Papaw (*Curly leaf*).—*Loc. cit.* (See Cacao.)

Passion vine (*Woodiness or bullet disease*).—NOBLE, R. J.: Woodiness of passion fruit. Cause of the disease discovered. *Agr. Gaz. New South Wales* **39**: 681–683. 1928. ——: Some observations on the woodiness or bullet disease of passion fruit. *Jour. & Proc. Roy. Soc. New South Wales* **62**: 79–98. 1928.

Pea (*Mosaic*).—DOOLITTLE, S. P. AND JONES, F. R.: The mosaic in the garden pea and other legumes. *Phytopath.* **15**: 763–772. 1925. (See also Legumes.)

Pea (*Streak*).—LINFORD, M. B.: Streak, a virus disease of peas transmitted by *Thrips tabaci. Phytopath.* **21**: 999. 1931.

Peach (*Little peach*).—(See special treatment, p. 273.)

Peach (*Phony disease*).—NEAL, D. C.: Phony peaches: a disease occurring in middle Georgia. *Phytopath.* **10**: 106–109. 1920. HUTCHINS, L. M.: Phony disease of the peach. *Jour. Econ. Ent.* **23**: 555–562. 1930.

Peach (*Rosette*).—(See special treatment, p. 276.)

Peach (*Yellows*).—(See special treatment, p. 265.)

Peanut (*Mosaic or rosette*).—STOREY, H. H. AND BOTTOMLEY, A. M.: The rosette disease of peanuts (*Arachis hypogœa* L.). *Ann. Appl. Biol.* **15**: 26-45. 1928.

Pelargonium.—(See Geranium.)

Peony (*Mosaic*).—COULSON, J. G.: Peony diseases. *Ann. Rept. Quebec Soc. Prot. Plants* **15** (1922–1923): 67–70. 1923.

Peony (*Ring spot*).—*Plant Dis. Rep. Supp.* **73**: 390. 1929.

Pepper (*Mosaic*).—BLODGETT, F. M.: A potato virus on peppers. *Phytopath.* **17**: 775–782. 1927. UPPAL, B. N.: Mosaic disease of chillies (Capsicum, in the Bombay Presidency. *Intern. Bul. Plant Protect.* **3**: 99. 1929. (See also Tobacco (Mosaic); Beet (Curly top).)

Pepper (*Infectious chlorosis*).—IKENO, S.: Studien über einen eigenthümlichen Fall der infektiösen Buntblätterigkeit bei *Capsicum annum. Planta Arch. Wiss. Bot.* **11**: 359–367. 1930.

Petunia (*Mosaic*).—ALLARD, H. A.: The mosaic disease of tomatoes and petunias. *Phytopath.* **6**: 328–335. 1916.

Petunia (*Ring spot*).—(See tobacco (Ring spot).)

Physalis (*Mosaic*).—(See Cucumber (Mosaic); also Beet (Curly top).)

Pineapple (*Yellow spot*).—ILLINGWORTH, J. F.: Yellow-spot disease of pineapples in Hawaii. *Phytopath.* **21**: 867–880. 1931. Also *Phytopath.* **21**: 999. 1931. Transmitted by *Thrips tabaci*. (See also Pea (Streak).)

Plantain (*Bunchy top*).—(See Banana (Bunchy top).)

Poinsettia (*Leaf curl*).—PAPE, H.: Eine neue Krankheit der Poinsettie. *Gartenw.* **31**: 772–773. 1927.

Pokeweed (*Mosaic*).—ALLARD, H. A.: The mosaic disease of *Phytolacca decandra. Phytopath.* **8**:51–54. 1918. (See also Cucumber (Mosaic) and Tobacco (Ring spot).)

Potato (*Leaf roll*).—(See special treatment, p. 301.)

Potato (*Mosaic*).—(See special treatment, p. 285. For other virus diseases of potato, see Potato Degeneration Diseases in General under Potato (Mosaic).)

Radish (*Mosaic*).—On rat-tail radish, *Raphanus sativus caudatus*. (See KULKARNI, G. S. under Okra.)

Raspberry (*Leaf curls* and *mosaics*).—Four varieties of leaf curls: (1) alpha type; (2) beta type; (3) severe streak; and (4) mild streak. Yellows is included under alpha and beta leaf curls. Eastern bluestem is included under severe and mild streak. Three types of mosaics: (1) red-raspberry mosaic; (2) yellow mosaic; and (3) mild mosaic. BENNETT, C. W.: Virus diseases of raspberries. *Mich. Agr. Exp. Sta. Tech. Bul.* **80**; 1–38. 1927. RANKIN, W. H.: Virus diseases of black raspberries. *New York (Geneva) Agr. Exp. Sta. Tech. Bul.* **175**: 1–24. 1931.

Rhododendron (*Mosaic*).—PAPE, H.: Mosaikkrankheit bei Rhododendron. *Gartenw.* **35**: 621. 1931.

Rhubarb (*Mosaic*).—DICKSON, B. T.: Mosaic of rhubarb. *Ann. Rept. Quebec Soc. Prot. Plants* **17**: 36–37. 1925.

Rice (*Stunt disease*).—TAKAMI, N.: Stunt disease of rice and *Nephotettix apicalis* M. *Jour. Agr. Soc. Japan* **241**: 22–30. 1901. HINO, I.: *Nogyo Oyobi Engei* **2**: 1223–1334. 1927.

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Rose (*Wilt or dieback*).—GRIEVE, B. J.: "Rose wilt" and "dieback." A virus disease of roses occurring in Australia. *Aust. Jour. Exp. Biol. Med. Sci.* **8**: 107–121. 1931.

Salsify (*Yellows*).—HASKELL, R. J. AND ARCHER, W. A.: Salsify yellows. *Plant Dis. Rep.* **13**: 139–140. 1929. Probably similar to aster yellows.

Sandal (*Spike disease*).—COLEMAN, L. C.: Spike disease of sandal. *Mysore Dept. Agr. Mycol. Ser. Bul.* **3**: 1–52. 1917. HART, W. C. AND RENGASWAMY, S.: Preliminary investigation into the cause and cure of the spike disease in sandal. *Ind. For.* **52**: 373–390. 1926. Conference on the spike disease of sandal, Bangalore, 1930. *Ind. For.* **57**: 215–233. 1931.

Sisal hemp (*Mosaic?*).—STANER, P.: Belgian Congo: a new disease of Sisal. *Intern. Bul. Plant. Prot.* **3**: 179. 1929.

Soy bean (*Mosaic*).—KENDRICK, J. B. AND GARDNER, M. W.: Soy-bean mosaic: seed transmission and effect on yield *Jour. Agr. Res.* **27**: 91–98. 1924.

Spinach (*Blight or mosaic*).—(See also Beet (Mosaic).) McCLINTOCK, J. A. AND SMITH, L. B.: The true nature of spinach blight and the relation of insects to its transmission. *Jour. Agr. Res.* **14**: 1–60. 1918. BÖNING, K.: Ueber die wechselseitige Uebertragbarkeit der Mosaikkrankheiten von Rübe und Spinat. *Centralbl. Bakt. Abt.* II. **71**: 490–497. 1927. HOGGAN, I. A.: Transmission of cucumber mosaic to spinach. *Phytopath.* **20**: 103–105. 1930.

Spinach (*Curl disease*).—BÖNING, K.: Ueber eine Blattdeformationskrankheit an Rübe und Spinat. *Zeitschr. Pflanzenkr.* **40**: 315–323. 1930. (See also Beet.)

Squash (*Curly top*).—McKAY, M. B. AND DYKSTRA, T. P.: Curly top of squash. *Phytopath.* **17**: 48. 1927. See also Beet (Curly top).)

Strawberry (*Witches' broom*).—ZELLER, S. M.: Preliminary study on witches' broom of strawberry. *Phytopath.* **17**: 329–335. 1927.

Strawberry (*Xanthosis*).—PLAKIDAS, A. G.: Strawberry xanthosis, a new insect-borne disease. *Jour. Agr. Res.* **35**: 1057–1090. 1927.

Strawberry (*Yellows*).—Claimed to be distinct from xanthosis. PLAKIDAS, A. G.: Report on strawberry virus disease project. *Plant Dis. Rep.* **13**: 129–131. 1929.

Sugar beet (*Curly top and mosaic*).—(See Beet.)

Sugar cane (*Fiji disease*).—LYON, H. L.: Three major cane diseases: mosaic, sereh and Fiji disease. *Hawaii Sugar Planters' Assoc. Exp. Sta. Bul.* Bot. Ser. 3: 1–43. 1921. Cell inclusions were named *Northiella sacchari*. WOOD, E. J. F.: Fiji disease in the Maryborough district. *Queensl. Agr. Jour.* **27**: 388–393. 1927. The cane leafhopper, *Perkinsiella sacchari*, thought to be the vector.

Sugar cane (*Mosaic, mottling or yellow-stripe disease*).—STEVENSON, J. A.: The mottling or yellow-stripe disease of sugar cane. *Jour. Porto Rico Dept. Agr.* **2**: 1–76. 1919. EARLE, F. S., *et al.*: Yellow-stripe disease investigations. *Jour. Porto Rico Dept. Agr.* **3**: 1–150. 1919. BRANDES, E. W. AND KLAPHAAK, P. J.: Cultivated and wild hosts of sugar-cane or grass mosaic. *Jour. Agr. Res.* **24**: 247–262. 1923. HAUSFORD, C. G. AND MURRAY, P. W. The mosaic disease of sugar cane and its control in Jamaica. *Jamaica Dept. Agr. Microbiol. Circ.* **6**: 1–39. 1926. REYES, G. M.: The mosaic disease of sugar cane. *Philippine Agr. Rev.* **20**: 187–228. 1927. TIMS, E. C. AND EDGERTON, C. W.: Behavior of mosaic in certain sugar-cane varieties in Louisiana. *Am. Jour. Bot.* **18**: 649–657. 1931.

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Sugar cane (*Streak*).—STOREY, H. H.: Streak disease of sugar cane. *Union S. Afr. Dept. Agr. Sci. Bul.* **39**: 1–30. 1925. —— AND McLEAN, A. P. D.: The transmission of streak disease between maize, sugar cane and wild grasses. *Ann. Appl. Biol.* **17**: 691–719. 1930. (See Corn (Streak).)

Sweet clover (*Ring spot*).—(See Tobacco (Ring spot).)

Sweet pea (*Mosaic*).—TAUBENHAUS, J. J.: Mosaic disease of the sweet pea. *Del. Agr. Exp. Sta. Bul.* **106**: 53–61. 1914. (See also MERKEL, L. under Legumes.)

Sweet potato (*Mosaic*).—ROSEN, H. R.: The mosaic disease of sweet potato. *Ark. Agr. Exp. Sta. Bul.* **167**: 1–10. 1920. ——: The mosaic disease of sweet potatoes with special reference to its transmissibility. *Ark. Agr. Exp. Sta. Bul.* **213**: 1–16. 1926. HARTER, L. I. AND WHITNEY, W. A.: Masking of sweet-potato mosaic. *Phytopath.* **19**: 933–942. 1929.

Tobacco (*Mosaic*).—Various local names have been used such as calico, brindle, mongrel, mottle top and string leaf for different manifestations of the symptoms. The following types of the mosaic virus have been recognized (Johnson, E. M., 1930): mild mosaic, types 1 and 2; severe mosaic, types 1 and 2; yellow mosaic; ring mosaic; and white mosaic. ALLARD, H. A.: Mosaic disease of tobacco. *U. S. Dept. Agr. Bul.* **40**: 1–33. 1914. GOLDSTEIN, BESSIE: A cytological study of the leaves and growing points of healthy and mosaic diseased tobacco plants. *Bul. Torr. Bot. Club* **53**: 499–599. 1926. JOHNSON, JAMES: The classification of plant viruses. *Wis. Agr. Exp. Sta. Res. Bul.* **76**: 1–15. 1927. JOHNSON, E. M.: Virus diseases of tobacco in Kentucky. *Kent. Agr Exp. Sta. Bul.* **306**: 289–415. 1930.

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Tobacco.—In addition to the true mosaics and ring spot, the following have been recognized on tobacco: Etch; etch +; severe etch; vein banding; coarse etch; cucumber types 1, 2 and 3; latent (healthy) potato virus; spot necrosis and other mixtures. See JOHNSON, E. M.: *Loc. cit.* VALLEAU, W. D. AND JOHNSON, E. M.: The relation of some tobacco viruses to potato degeneration. *Kent. Agr. Exp. Sta. Bul.* **309**: 475–507. 1930. (See also BURNETT, G. AND JONES, L. K. under Tomato (Streak).)

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Tobacco (*Rotterdam-B disease*).—JOCHEMS, S. C. J.: Handleiding voor de herkenning en bestrijding van de ziekten van Deli-Tabak. *Meded. Deli Proefsta. te Medan-Sumatra.* Ser. II. **43**: 1–39. 1926.

Tobacco (*Vein streak*).—JOCHEMS, S. C. J.: Twee neuwe virusziekten bij Deli-TABAK. *Bull. Deli Proefstat. te Medan-Sumatra* **30**: 1–24. 1930. Possibly same as Streifenkrankheit (BÖNING, K. *Loc. cit.*).

Tobacco (*Witches' broom*).—YOUNG, P. A.: Tobacco witches' broom. *Am. Jour. Bot.* **16**: 277–279. 1929.

Tomato (*Aucuba mosaic*).—BEWLEY, W. F. AND BOLAS, B. J.: Aucuba or yellow mosaic of the tomato plant: reaction of infected juice. *Nature* **125**: 130. 1930.

Tomato (*Tobacco mosaic*).—ALLARD, H. A.: The mosaic disease of tomatoes and petunias. *Phytopath.* **6**: 328–335. 1916. GARDNER, M. W. AND KENDRICK, J. B.: Tomato mosaic. *Ind. Agr. Exp. Sta. Bul.* **261**: 1–24. 1922. JOHNSON, E. M.: *Loc. cit.* BURNETT, G. AND JONES, L. K.: *Loc. cit.*

Tomato.—In addition to the true mosaics, the tomato is susceptible to the other tobacco viruses except ring spot. JOHNSON, E. M.: *Loc. cit.* BURNETT, G. AND JONES, L. K.: *Loc. cit.*

Tomato (*Streak*).—VANTERPOOL, T. C.: The stripe or streak disease of tomatoes in Quebec. *Ann. Rept. Quebec Soc. Prot. Plants* **16**: 116–123. 1924. VALLEAU, W. D. AND JOHNSON, E. M.: Some possible causes of streak in tomatoes. *Phytopath* **20**: 831–839. 1930. BURNETT, G. AND JONES, L. K.: The effect of certain

potato and tobacco viruses on tomato plants. *Wash. Agr. Exp. Sta. Bul.* **259:** 1–37. 1931.

Tomato (*Fern leaf*).—MOGENDORFF, N.: "Fern leaf" of tomato. *Phytopath.* **20:** 25–46. 1930. This disease is caused by *Cucumber virus* 1 and is readily transmitted by the peach aphid (*Myzus persicæ*).

Tomato (*Leaf roll*).—DYKSTRA, T. P.: Leaf-roll transmission from potato to other solanaceous plants by means of *Myzus persicæ*. *Phytopath.* **20:** 883. 1930.

Tomato (*Psyllid yellows*).—BINKLEY, A. M.: Transmission studies with the new psyllid-yellows disease of solanaceous plants. *Science*, n. s., **70:** 115. 1929. (See also Potato.)

Tomato (*Spotted wilt*).—SAMUEL, G., BALD, J. G. AND PITTMAN, H. A.: Investigations on "spotted wilt" of tomatoes. *Council Sci. Ind. Res. Austr. Bul.* **44:** 1–48. 1930. Recorded also in Wisconsin. *Phytopath.* **21:** 106. 1931.

Tomato (*Western blight, yellows or curly top*).—SEVERIN, H. H. P.: Transmission of tomato yellows or curly top of the sugar beet by *Eutettix tenellus* Baker. *Hilgardia* **3:** 251–274. 1928. SHAPOVALOV, M.: Experiments on the control of tomato yellows. *U. S. Dept. Agr. Tech. Bul.* **189:** 1–23. 1930. First definitely connected with curly top by McKay and Dykstra (*Phytopath.* **17:** 39. 1927).

Tomato (*Witches' broom*).—YOUNG, P. A. AND MORRIS, H. E.: Witches' broom of potatoes and tomatoes. *Jour. Agr. Res.* **36:** 835–854. 1928.

Tulip (*Breaking*).—CAYLEY, D. M.: "Breaking" in tulips. *Ann. Appl. Biol.* **15:** 529–539. 1928. HUGHES, A. W. M.: Aphids as vectors of "breaking" of tulips. *Ann. Appl. Biol.* **18:** 16–29. 1931.

Turnip (*Mosaic*).—GARDNER, M. W. AND KENDRICK, J. B.: Turnip mosaic. *Jour. Agr. Res.* **22:** 123–124. 1921. (See also Crucifers.)

Wheat (*Mosaic or rosette*).—(See special treatment, p. 277.)

SECTION IV

PARASITIC DISEASES

CHAPTER XIII

BACTERIAL DISEASES OF PLANTS

Previous to 1878 the occurrence of bacterial diseases of plants had not been demonstrated, although bacteria had already been accepted as important pathogenes for man and domestic animals. Our knowledge of bacterial diseases of plants is therefore the result of studies made within the last 45 years, but the most rapid advancement has been made during the last 20 years.

Early Knowledge of Bacterial Diseases of Plants.—It is significant that the minds of investigators in widely separated parts of the world were turned to the possibility of bacterial invasion of plants, as evidenced by the proof of the occurrence of bacterial diseases of plants nearly simultaneously in America, France and Holland. This historical feature may be shown in the following tabulation:

Name of disease	Host	Investigator	Years of work	Country
Fire blight..............	Pear and apple	Burrill	1878–1883	Illinois, U. S. A.
Rose-red disease.........	Wheat kernels	Prillieux	1879	France
Yellow disease........ ..	Hyacinths	Wakker	1883–1889	Netherlands

It is also worthy of note that Comes in Italy in 1880 recognized the pathogenicity of bacteria for plants, although less specific diseases were described. It is interesting to note that Woronin discovered bacteria in the root tubercles of legumes (lupines) in 1866, 12 years previous to the important work of Burrill. Notwithstanding these early proofs of specific bacterial diseases, workers, especially in Germany, were slow to accept the findings of their contemporaries. Sorauer, the noted German plant pathologist, accepted the findings of Wakker as early as 1886. As late as 1897, the eminent German bacteriologist, Dr. Alfred Fischer, denied the occurrence of specific bacterial diseases of plants. He argued that bacteria could enter a plant only through wounds and that their development would soon be checked by the formation of an excluding cork layer; further that stomatal infection was altogether impossible. His claims were again put forward 2 years later and they were also supported by Wehmer, another German scientist. The claims of these Germans called forth a critical discussion of the evidence in support of bacterial diseases of plants by Dr. Erwin F. Smith, of the U. S. Department of Agriculture in 1899 and in 1901. Since that time no worker has appeared to dispute the importance of bacteria as plant pathogenes, and the number of specific bacterial diseases known to science has rapidly increased. The

publication by Smith of the first volume of "Bacteria in Relation to Plant Diseases in 1905," with the later volumes of this monumental work, served as a stimulus to the investigations that have followed.

The Number of Bacterial Diseases of Plants.—In 1896 Smith stated that "there are in all probability as many bacterial diseases of plants as of animals." In 1920 the same authority reported the occurrence of specific diseases on hosts scattered through more than 150 genera and over 60 families. These include three diseases of Gymnosperms, 23 of Monocots, with the balance in the various Dicot families from the willows to the Composites. Many of the hosts are important cultivated plants or wild plants of economic importance. According to the opinion of Smith, "It appears likely that eventually bacterial diseases will be found in every family of plants from the lowest to the highest." Each year since the publication of the statements quoted has witnessed numerous additions to the long list of recognized bacterial diseases of plants. In the recent "Manual of Determinative Bacteriology" (Bergey, 1930) the plant pathogenes are placed in two genera, Erwinia with 12 species and Phytomonas with 81 species. This does not represent the full number of bacterial plant pathogenes, since many others have been described. Elliott (1930) gives descriptions of 13 Aplanobacter, 53 Bacillus, and 111 Bacterium.

The General Morphology of the Bacteria.—These minute organisms are probably the simplest of our non-chlorophyll-bearing plants and include a large number of distinct species which can be recognized partly on the basis of structural characters and partly from their physiological or cultural characters. Mode of life has had but little effect on their structural characters, the plant body being essentially the same, whether they live as scavengers on dead refuse or inorganic food materials or adopt the rôle of robbers and live at the expense of other organisms, plant or animal, as parasites.

The plant body in its simplest form in the true bacteria (Eubacteriales), sometimes called the *lower bacteria*, consists of a single cell, which can be assigned to one of three general types: (1) globular or spherical forms, the *coccus type;* (2) short to long cylindrical or rod-shaped, the *bacillus type;* and (3) the short or long, spiral, cylindrical forms, the *spirillum type.* The cells of the different types may be held together as pairs, chains, long or short filaments, long spirals or, in the coccus forms, as packets or cell masses, but in every case the single cell is to be considered as the individual, and the various groupings as aggregates of individuals. Under certain conditions irregular or branched forms may be assumed. The bacteria vary in size from coccus forms 0.15 to 1 micron in diameter to bacillus or spirillum forms 0.3 to 3 microns in diameter and 1 to 6 or more microns in length.

Each cell is surrounded by a definite cell wall or membrane, which is nitrogenous in chemical character, rather than a carbon compound (cellulose of fungi and higher plants). It is a common thing for the wall to undergo a mucilaginous modification, hence groups of bacteria are viscid or slimy and may be held together as sheathed filaments, irregular masses or thin, scum-like sheets. The living substance, the protoplasm, shows but little differentiation. Certain species of bacteria are always

inactive or incapable of locomotion, while others under certain conditions are able to swim actively through a liquid medium, or have the power of

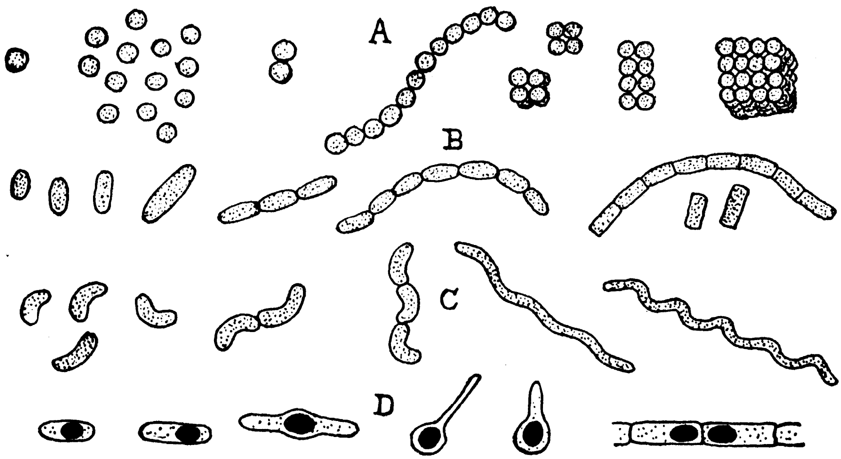


FIG. 86.—Diagram illustrating the morphology of the true bacteria. *A*, coccus forms; *B*, bacillus forms; *C*, spirillum forms; *D*, types of spore formation.

locomotion. This motility is the result of the lashing movements of delicate vibratile threads, the *flagella*, varying in number and distribution.

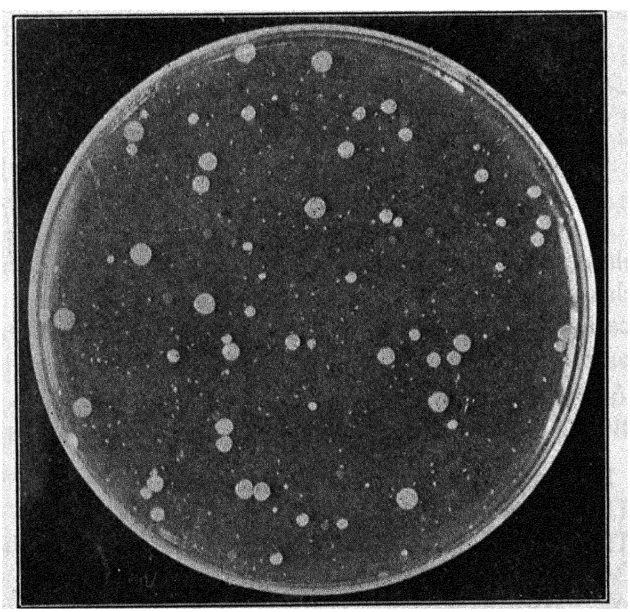


FIG. 87.—Poured-plate isolation showing colonies of bacteria obtained from a bacterial blight of Antwerp raspberries.

Reproduction is by simple fission or cell division, hence the common name, the *fission fungi*. This habit also suggested the scientific name, the Schizomycetes, which comes from Greek words meaning splitting

fungi. Under suitable conditions for growth a separation of a single cell into two will be completed in 20 to 30 minutes, but this rate of multiplication is not kept up for long periods because of the operation of various unfavorable factors. Certain species of bacteria are able to form specialized reproductive bodies or spores. The most general form, the *endospore*, is formed by the contraction and the concentration of the protoplasmic body, which assumes a globular or oval form and surrounds itself with a firm, solid membrane, while the old surrounding cell membrane will ultimately disappear. Löhnis describes four other types of reproductive bodies. The endospores are much more resistant to desiccation, heat or other unfavorable factors than the vegetative cells, and under suitable conditions will germinate and produce new vegetative cells. None of our plant pathogenes are known to produce endospores.

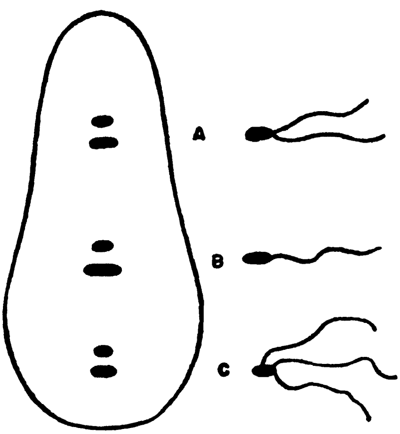


FIG. 88.—Comparative size of apple-scab spore and three species of bacteria. *A*, crown-gall bacteria; *B*, bacteria of black rot of cabbage; *C*, fire-blight bacteria.

The plant pathologist is interested in one of the groups of the higher bacteria, the Actinomycetales. In this group the plant body is filamentous, often branched and quite fungus-like in character, and sometimes reproduces by means of specialized spores or conidia. Some authorities refer these organisms to the simple imperfect fungi, the Hyphomycetes, rather than to the Schizomycetes. The other groups of bacteria with which the plant pathologist is not concerned are presented in the following outline of classification.

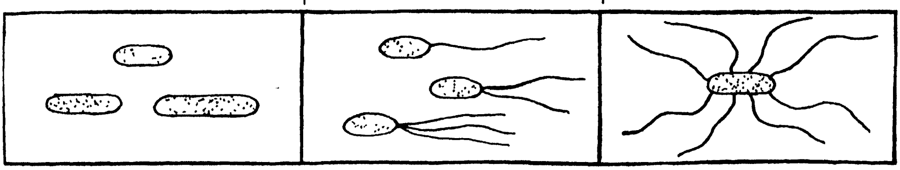
Classification of Bacteria in General and of Bacterial Plant Pathogenes.—According to the recent classification by the Committee of the Society of American Bacteriologists (Bergey's Manual, 1923, 1930), the Schizomycetes or bacteria are arranged in the following six orders:

Order I. Eubacteriales, the true bacteria.
Order II. Actinomycetales, the mold-like bacteria.
Order III. Chlamydobacteriales, the alga-like iron bacteria.
Order IV. Thiobacteriales, the alga-like sulphur bacteria.
Order V. Myxobacteriales, the myxobacteria.
Order VI. Spirochætales, the protozoan-like bacteria or Spirochætes.

The forms that are of phytopathological importance belong, for the most part, to the family of the Bacteriaceæ of the true bacteria, while the genus Actinomyces of the Actinomycetales furnishes at least one plant

pathogene, *Actinomyces scabies,* the cause of the common scab of the Irish potato.

It seems strange that coccus and spirillum forms of the true bacteria are of little or no importance as plant pathogenes, and that even among the rod forms none of the spore formers has parasitized plants. For some years plant pathologists generally accepted the classification of Migula, but Smith made a departure from this by substituting Bacterium for Pseudomonas and establishing a new generic name, Aplanobacter, for the non-motile forms referred to Bacterium by Migula, while a greater change has been made by the Committee of the Society of American Bacteriologists. These three different classifications now appearing in current American literature may be the cause of some confusion. In order to make them plain as they apply to plant pathogenes, they are presented in the following tabular comparison:



Non-motile	Motile	Motile
Bacterium: Migula Aplanobacter: Smith	Pseudomonas: Migula Bacterium: Smith	Bacillus: Migula Bacillus: Smith Erwinia: S. A. B.
Phytomonas: S. A. B.		

It could not be expected that such simple forms as the bacteria could be assigned to species on the basis of morphological characters alone. They must be still further differentiated by their cultural characters on various artificial media, by their physiological behavior and by their pathogenicity. For this reason the student of plant pathology who would make real progress in the study of bacterial diseases of plants must be well grounded in the fundamentals of bacteriological technique.

Types of Bacterial Diseases.—Three types of bacterial disease may be recognized:

1. *Vascular diseases* are characterized by primary invasions of the water-conducting vessels of the fibrovascular bundles by the bacterial pathogene. In diseases of this type the water-conducting vessels may become so filled with the bacteria that water can no longer be supplied to the foliage and the plant wilts rather suddenly. This is well illustrated in the *wilt* of cucurbits due to *Bacillus tracheiphilus* E.F.S. If stems of such affected plants are cut across, a white, viscid, bacterial ooze will appear from the ends of the xylem portions of the vascular bundles. The *brown rot* (*Pseudomonas solanacearum*), which affects the potato, eggplant,

tomato, tobacco and other species of the nightshade family in the southeastern United States, is a parenchymovascular disease of great importance. In this disease the invaded vessels are stained brown or black and sudden wilting results, but the pathogene invades adjacent tissues of parenchymatous character and causes the destruction of pith and cortex with the formation of bacterial cavities. The *black rot* (*P. campestris*) of the cabbage, cauliflower and other members of the mustard family is a vascular disease in which wilting is a less evident symptom than in many other vascular invasions. The primary invasion is through the vessels of the leaf veins, which are turned brown as the bacteria advance downward and inward. In this way the bacteria may reach the center of a cabbage head and spread to adjacent portions, and finally an offensive-smelling, soft rot may result from the action of secondary invaders of a saprophytic nature.

2. *Parenchyma diseases* are troubles in which the pathogene invades the soft or succulent parenchyma tissues of the host, as a primary feature,

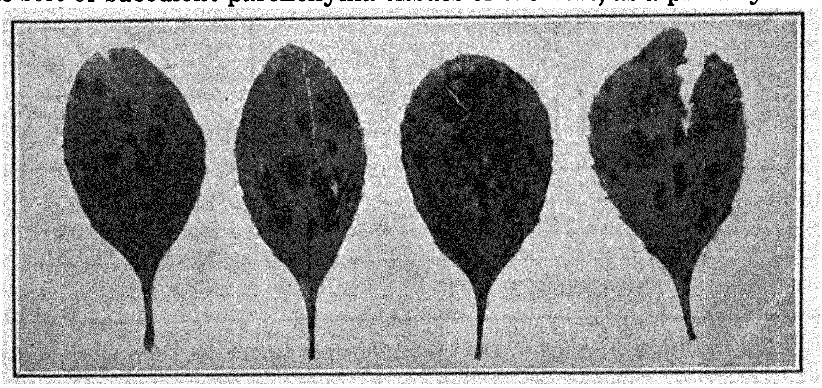


FIG. 89.—Bacterial leaf spot of the common barberry.

but may sometimes break into adjacent vascular elements. In such case there is a necrosis of tissue without hyperplasia, and a spotting, blighting or rotting of the affected parts is the final result. The fire blight of the apple and pear is an outstanding illustration of a disease of this type. The soft rot (*Bacillus carotovorus* Jones) of carrots and many other fleshy roots, stems, rhizomes or fruits is a rapid, soft and wet rot of parenchyma tissues that are filled with reserve food. The blackleg, basal stem rot or bacterial black rot of the potato is a parenchyma disease in which the bacteria invade the cortex of the basal portion of the stem, producing the blackened and shriveled stem bases which suggest the common name "blackleg." The bacteria often infect the tubers and cause a black rot or decay. The angular leaf spot (*Pseudomonas malvacearum* Smith) is an illustration of a somewhat different type of parenchyma disease, which first appears on the leaves as water-soaked spots, which finally turn to brown, angular areas. It affects the stem also,

producing the "black arm" or gummosis and the capsule causing a "boll rot."

3. *Hyperplastic diseases* are characterized by tubercle, tumor or gall formation or by the development of additional organs (shoots or roots) from adventitious, dormant or latent buds. In diseases of this type the bacteria stimulate certain cells to increased activity and as a result of this abnormal cell division the structures noted above are developed. The well-known crown gall (*P. tumefaciens* Smith and Townsend) of fruit trees, shade trees and many other plants is a striking illustration of a hyperplastic overgrowth which may develop on various organs. The hairy root of the apple and other trees due to *P. rhizogenes* Riker *et al.* illustrates another type of overgrowth which is characterized by the production of an excessive number of small fibrous roots, generally without tumor or gall formation. Mention may also be made of the olive knot or tubercle (*P. savastanoi* Smith), which appears as irregular knots or excrescences of a spongy or cheesy character on aerial parts, especially on trunk or branches.

How Bacteria Invade Their Hosts.—The external surface of the plant body of seed plants is covered in large part by an epidermis with an external cuticle or also with cuticularized external walls, or by a more impervious layer of cork cells, the periderm. Some parasitic fungi are able to make their way through such unbroken epidermal walls, but pathogenic bacteria seem unable to penetrate cuticularized walls or layers of cork cells. This leaves wounds, natural openings or surfaces unprotected by an external cuticle as the possible avenues of entrance.

1. *Entrance through Wounds.*—It was at first thought that bacteria could enter plants in no other way than through wounds, but today this way of infection is known to be but one of several of considerable importance. Mechanical injuries of various kinds which bruise or break the tissue subject the injured organs to invasion by wound parasites. The cell sap and the protoplasmic contents from the injured cells offer a food or culture medium in which bacteria may be lodged and in which they can make their initial development. By repeated fission a mass consisting of many bacteria may result and adjacent uninjured cells may be affected as the bacteria work their way into the deeper tissues. A few specific examples will suffice to emphasize the entrance of bacteria through wounds. It has frequently been observed that the olive-knot organism gains entrance to twigs through hail bruises; wounds made in grafting or budding constitute an important place of infection for the crown-gall bacteria, and such wounds will explain the common occurrence of crown-gall tumors at a point just below the ground level in many fruit trees; the soft rots of potatoes, carrots and other root crops follow mechanical injuries or the inroads of fungous parasites; injuries to the root system of Solanaceous plants by cultural operations or the feeding of nematodes or

insects are frequently responsible for attacks of bacterial wilt; feeding of the striped cucumber beetles may be responsible for the infection of cucumbers with bacterial wilt, while bark-boring beetles or sucking insects may make tunnels or feeding punctures in apples, pears and other hosts in which the bacteria of fire blight first establish themselves. It seems probable that guttation ruptures of young leaves, especially of cereals, may make openings through which infection may result.

2. *Entrance through Stomata.*—These natural openings, which are so numerous on most green structures, constitute passageways leading into the system of intercellular spaces which permeate the tissues. With so many gateways it seems strange that stomatal invasion is not more frequent. The way in which a stomatal invasion may take place may be

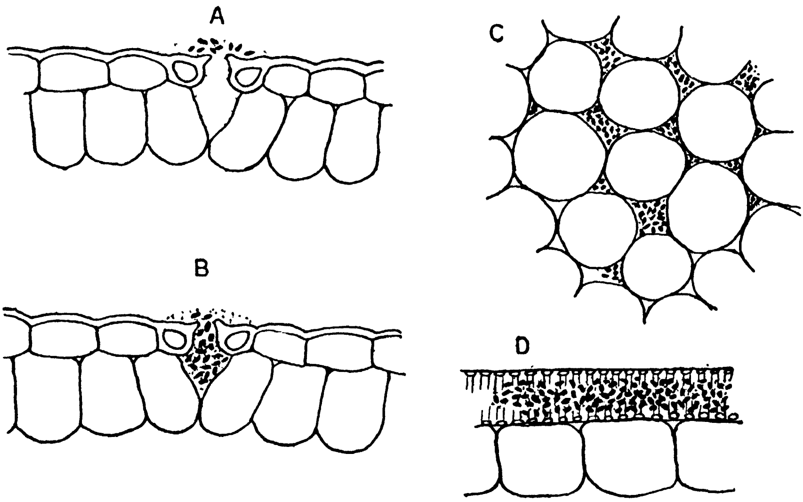


Fig. 90.—*A*, portion of a leaf section showing bacteria in water film above a stoma; *B*, penetration of the bacteria into the substomatal chamber; *C*, bacteria in the intercellular spaces of a parenchyma tissue; *D*, bacteria advancing in a spiral vessel. All semidiagrammatic.

briefly outlined. Suppose a single bacterium of a species pathogenic for the host finds lodgment in a film of water over a stomatal opening. Under favorable conditions cell division follows in rapid succession. The resulting bacteria are pushed or work their way into the stomatal opening and soon come to fill the substomatal chamber. They increase in numbers until they push into the adjacent intercellular spaces and a watery, translucent spot results, while with further action the cells of the lesion may be killed and a leaf spot or a spreading lesion may result. Since the first proof of stomatal invasion in the black spot (*Pseudomonas pruni* Smith) of the plum by Smith in 1902–1904, numerous cases of stomatal infection have been studied. This type of invasion has repeatedly been demonstrated by spraying pure cultures of pathogenes upon the surface of perfectly sound plants. It is therefore well established

that perfectly normal structures under the moisture conditions which frequently prevail in nature may become infected with a bacterial pathogene.

3. *Entrance through Water Pores.*—These structures, which are really specially modified stomata located in groups at the margins of certain leaves over the ends of veins, seem also well suited to the entrance of bacteria, since liquid moisture accumulates at such points during the periods of retarded evaporation. The most noteworthy case of water-pore invasion is to be found in the black rot of the cabbage. The bacteria develop first in the tissue immediately below the water pores, but soon make their way into the spiral vessels of the vein terminal and then advance down the vessels as cell division proceeds, rapidly producing the characteristic black venation.

4. *Entrance through Nectaries.*—The nectar-secreting glands of flowers offer another possible place of entrance, since the glandular tissue has no protection of cuticle or cuticularized layers and the sweetish nectar presents a food that is favorable for the growth of bacteria. Certainly, the anatomical features and the presence of the nectar are favorable, hence the infrequency of bacterial invasions of flowers by this portal must be ascribed to the lack of proper chemical relations of host tissue and possible pathogenes. In 1891, Waite first demonstrated the relation of bacteria of fire blight to the blossom-blight phase of that disease, and proved that the bacteria are introduced into the nectar by the visits of bees or other nectar-seeking insects. The bacteria left by visiting insects multiply with wonderful rapidity in the accumulated nectar, soon invade the nectaries through their unprotected glandular surface and then migrate downward into the ovary, flower pedicel and even into the stem, causing the characteristic blighting of flowers and fruit spurs. There are but few other proved cases of nectarial infection, but it seems probable that other examples will be discovered as more emphasis is given to the study of bacterial diseases. Extra-floral nectaries and the stigmas of flowers offer additional unprotected surfaces, but no bacterial pathogenes are known to enter by those channels.

5. *Entrance through Lenticels.*—The extent to which bacteria can enter through these passageways which lead through the corky bark into the underlying parenchyma tissue of twigs, woody branches or modified stems is uncertain. This seems to be a possible way of entrance of bacteria into potato tubers, especially those which cause soft rot. The possibility of lenticellate infection is one worthy of more detailed investigation.

The Location of Bacteria in Diseased Tissue.—The majority of bacteria which cause disease in plants enter into the tissues of various organs or parts by some of the methods as outlined above, but in a few cases diseases are caused by the development of the pathogene between certain closely appressed organs. This *superficial position* of the bacteria

is illustrated in Rathay's disease of orchard grass, in which the organisms are found between the glumes of the affected heads, and the gum-bud disease of carnations, in which the bacteria accumulate between the outer petals of unopened buds. Diseases similar to that of orchard grass have been described by O'Gara from wheat grass in Utah and by Hutchinson from wheat in India.

Bacteria which actually penetrate the tissues may be: (1) *intercellular*, or in the spaces between cells, as in most of the parenchyma diseases; (2) *intravascular*, or in the water-conducting vessels of the xylem, as in the wilts and other vascular diseases; and (3) *intracellular*, or within the interior of cells. In invasions which are primarily intercellular or during the first stages of intercellular infection, the bacteria may enter the vessels to a limited extent. In many vascular invasions, the bacteria later break out into the surrounding parenchyma tissue and form intercellular pockets, while in advanced decompositions of tissue, bacteria may enter the dead cells and assist in the work of destruction. In a few cases, however, the bacteria gain access to the interior of living cells at the very beginning. This would seem to imply the ability of the pathogene to penetrate unbroken walls of cells by its own activities, possibly by digestive action and perhaps by entering minute wall perforations. Smith states "that the crown-gall organism occurs within the rapidly dividing cells" (1911), but that it does not seem to be very abundant in the affected tissues, but recent investigations by Riker and others seem to oppose this idea (see Crown Gall). The symbiotic bacteria of the root tubercles of legumes offer an example of a copious intracellular development, with the bacteria persisting until the tubercles are matured and disintegrated.

The Action of Bacteria on Their Hosts.—There may be a

. . . mechanical splitting, tearing or crushing due to the enormous multiplication of the bacteria within confined spaces. The whole intercellular mechanism of soft plants may be honeycombed and flooded in this way, and if the cavities are near the surface the tissue may be lifted up or the bacteria may be forced to the surface through lenticels or stomata in the form of tiny beads or threads, or by a splitting process (Smith).

The principal actions are of a chemical nature, some of the most important being: (1) The *separation of cells* from each other by the digestive action of enzymes upon the pectic substances of the middle lamellæ, leading to the production of cavities in various tissues. (2) The production of enzymes which convert starches into sugars, complex sugars into simpler forms or digest and make possible the assimilation of proteids and other nitrogenous compounds. (3) The formation of injurious acids, alkalis or toxic substances of some other character as by-products of their activity, which either inhibit physiological processes or actually kill the protoplasm by their poisonous action. Such chemical changes may

result in necrosis of tissue or the blighting of organs, or dry or soft rots may follow. (4) The production of substances which stimulate cells to abnormal activity rather than depressing their life processes or killing them outright.

The Reaction of the Host.—In every invasion by a pathogenic form there must be a contest between the host tissue and the intruder. It is possible that in many incipient infections the forces of the host win the contest very early in the struggle and consequently prevent the formation of visible lesions. It is only when the resisting powers of the host tissue are overbalanced by the aggressions of the parasite that disease becomes evident. If it were not for host resistance, parasites when once established would always multiply indefinitely until a fatal ending would curtail their activities, but we know that many bacterial lesions are of limited extent and that finally the intruding organisms die out and disappear, leaving but the marks of the battle, while the host still lives. There is but little evidence that parasitized plants develop compounds comparable to the antibodies which are formed in infectious diseases of animals; at least repeated attacks by a bacterial pathogene confer no immunity upon the host plant. It is certain, however, that bacterial development is frequently checked, but this may be due to the products of host metabolism on the one hand, or to the by-products of bacterial metabolism on the other.

In addition to the killing effect upon host tissues which results in the death of localized areas, entire organs or parts, or the entire plant, profound changes or modifications of plants invaded by bacterial pathogenes may result. Some of the more striking effects are *retarded or lessened growth* of certain groups of tissues, certain parts or organs or the entire plant; *changes of color*, involving conservation of chlorophyll, with the development of a deeper green or the change to yellow or some other color; *distortions* of leaves, stems or other parts not due to hypertrophies or to hyperplastic changes; and finally the *development of new tissue*, new organs or abnormal overgrowths as a result of either *hyperplasia*, or increased cell division, or of the hypertrophy of cells.

In some of the simple bacterial attacks the advance of the bacteria in the tissues may be checked by the construction of a barrier of cork cells in advance of the invaded tissues. Cells which normally would remain inactive are stimulated to cell division and produce a cork cambium which builds the impervious wall of cork cells. In other cases the increased cell activity, expressed in hyperplasia, may be so directed as to cause the development of organs of normal structure, roots or shoots, but either out of place or in excessive numbers (hairy root and witches' brooms).

In other cases of hyperplastic response cell division runs riot, unchecked and undirected, with the result that irregular or formless

overgrowths, cankers, tubercles, tumors or galls are formed. These may be illustrated by the crown gall on both woody and herbaceous hosts, the olive knot, the tuberculosis of the oleander, the galls of several species of pines, citrus canker and other less perfectly known canker diseases (poplar, oak). In these cases it is probable that some chemical substance produced by the bacteria or by the irritated cells excites the cells to cell division, which proceeds with great rapidity. In many of the overgrowths the cells divide so rapidly that they remain small, and little differentiation takes place, with reduction of vascular elements. In other cases the same gall or tumor may exhibit hyperplasia and also enlargement or hypertrophy of some of the cells.

The Dissemination of Bacterial Diseases.—Bacterial diseases may be introduced into a new environment or spread from diseased to healthy plants in a great variety of ways. Diseased plants may harbor the disease and serve as centers of infection when transported to distant points or when allowed to remain in fields or orchards. Some of the more important agents of transmission may be mentioned: (1) *Seed* used in the broadest sense to include true seeds, fruits, bulbs, tubers or other propagating stock. The black rot of cabbage, Stewart's wilt disease of sweet corn, black chaff of wheat, yellow disease of hyacinths, and blackleg of potato are recognized seed-borne bacterial diseases. (2) *Insects and other animal life,* including birds, mollusks and worms. Here may be mentioned the insect transmission of fire blight of apple, pear and other hosts (by various sucking and chewing insects) and the wilt of cucurbits (by striped cucumber beetles, *Diabrotica spp.*); the transmission of the olive tubercle by snails; and the work of root-knot nematodes or round worms, which open the way for the entrance of the bacterial wilt or brown rot of tobacco, tomato and other susceptible hosts. (3) *Contaminated fertilizer.* The compost heap may harbor pathogenes which are later introduced into the field. Scab and rots of potato, black rot of cabbage and other troubles may be disseminated in this way.

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BLACK ROT OF CRUCIFERS

Pseudomonas campestris (Pam.) E. F. S.

The black rot of the cabbage and other crucifers is a vascular bacterial disease which causes dwarfing and rotting of plants and spotting or blighting of leaves and in extreme cases death of the host. The disease has been referred to as a bacteriosis, bacterial rot, brown rot or, most frequently, as the black rot.

History.—The disease was first recognized by Garman in Kentucky in 1890, but its bacterial nature was not proved. The infectious nature of the disease was demonstrated by Pammel in 1893–1895 by his study of a bacteriosis of rutabagas in Iowa, but he did not associate the trouble with the disease on cabbage and other hosts which was studied later by Russell and Harding and by E. F. Smith. These investigators, working independently, arrived at essentially the same results, the work of Smith being published a year earlier (1897) than that of the others. Both papers extended the work of Pammel and reported the infections of the leaves through water pores. Important later contributions were made in publications of Harding (1900), Smith (1898, 1903, 1911), Stewart and Harding (1903), and by Harding, Stewart and Prucha (1904). These workers demonstrated the ineffectiveness of the removal of diseased leaves as a method of control and showed that the causal bacteria are frequently carried on the seed. It is interesting to note that Smith used illustrations of black rot in his dispute with Fischer concerning the occurrence of bacterial diseases. In 1902–1903, Brenner, a student of Fischer's, confirmed the work of Smith that had been disputed by Fischer. The demonstration of the prevalence of the disease in Europe by Harding (1900) stimulated the researches of Van Hall (1900) and Hecke (1901–1902). Mention should be made of recent work on cotyledon infection by Drechsler (1919), the study of the disease on Chinese cabbage by Brown and Harvey (1920), the description of variable effects on cauliflower by Clayton (1924), the demonstration of the effectiveness of the hot-water treatment of seed by Walker (1924) and the importance of seed-bed sterilization in lessening the disease by Clayton (1924).

Geographic Distribution.—"Black rot has been reported from practically all states east of the Mississippi River and from several west of it, especially from Iowa and Nebraska. For 20 years or more it has been destructive in the older cabbage-growing sections of the country, especially in the states of Ohio, Wisconsin, Michigan, New York, Iowa, Indiana and Pennsylvania. In more recent years several other states, particularly Maryland, Virginia, New Jersey and Texas, have reported serious outbreaks. The disease has been injurious to cabbage as far south as Florida and extends through all the states north into Canada. In 1908 it was reported from the state of Washington, but with a few possible exceptions it is of rare occurrence in the Rocky Mountain and Pacific Coast states. It is also well known in almost all parts of Europe and has been reported from the islands of Cuba, Porto Rico, New Zealand and the

Philippines" (Harter, Jones and Walker, 1923). Harding confirmed the report of Paddock that the black rot was destructive in Colorado in 1901 and 1902, and further attention was given to the disease in that state by Sackett in 1909. In 1911, Smith and Smith reported that "the trouble is very uncommon in California if it occurs at all."

Symptoms and Effects.—The earliest symptom of the disease, especially in the cabbage, is generally a yellowing of the foliage with a blackening of the veins, beginning at the leaf margin or around some insect injury, with a progressive development of the stain downward into the petiole and then into the vascular elements of the main stem.

When once it has established itself in the main stalk it spreads rapidly through the whole plant. Traveling as it does through the veins and bundles of the plant, it seriously interferes with the channels by which water is distributed to the

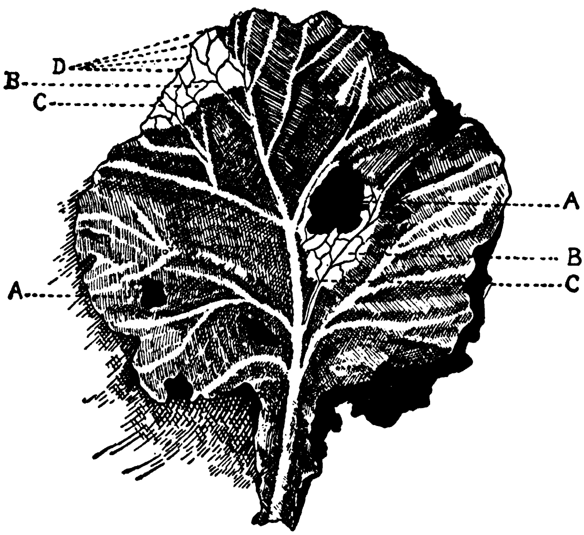

FIG. 91.—Cabbage leaf showing manner of infection. *A*, holes eaten by insects; *B*, diseased areas unshaded except blackened mesh of veinlets; *C*, blackened veinlets affected by disease; *D*, water pores through which the black-rot bacteria enter to produce a marginal infection. (*After Russell, Wis. Bul.* **68.**)

tissues of the leaf. This, combined with the actual invasion of the leaf tissue with the specific cause of the disease, causes the leaves to wilt, turn yellow, dry up and become thin and parchment-like in texture. The veins and fibrous strands that course through the plant are usually blackened, and often these alone are affected, the contiguous succulent tissues remaining apparently unchanged for a time at least (Russell).

Affected leaves are not wet or decayed, and in severe cases there may be a gradual shedding of the lower leaves, finally resulting in a long bare stem marked only by the leaf scars and frequently with shoots pushing out from the axillary buds, while a terminal tuft of more or less distorted leaves crowns the stem. Dwarfing is one of the common effects of the

disease and frequently one side of the plant is more affected than the other. Early attacks may destroy the young plant or it may make a crippled development without the formation of a head. The disease may infect seedlings in the seed bed or the attacks may come only after the plants have been transplanted to the field. Seedling infections may develop even before the unfolding of the first leaf, showing a characteristic charring of the tissue along the edge of the cotyledon sinus. This initial symptom may be followed by the collapse of the cotyledons and the death of the seedling.

One of the most characteristic features of the disease is the blackening of the vascular elements. A cross-section of the fleshy petiole of an affected leaf may show all the bundles brown or black, and a cross-section of the stem, if the disease is sufficiently advanced, will show a brown or black woody cylinder. In fleshy structures, as in roots of turnips or in the edible portion of kohlrabi, pronounced cavities may be hollowed out in the succulent parenchyma. In certain cases a yellowish slime will ooze out from the cut ends of the vascular bundles—for example, in slices of infected turnip roots.

A deviation from the symptomology as described for the cabbage may be noted for the cauliflower. The infections occur over the entire leaf instead of along the margins and in dry weather may dry up and cause leaf perforations or in wet weather may spread and cause a wet stump rot. Lesions also may be formed on the seed pods. Numerous leaf-spot infections are also characteristic of the trouble on the Chinese cabbage.

In black rot the sequelæ are even more serious than the disease itself, for the affected plants are frequently invaded by bacteria which cause a destructive soft rot that may spread throughout the head and transform it into a black, slimy, foul-smelling mass. Plants affected by the primary disease alone have no conspicuous odor. The losses are not confined to the field but are continued into storage. In the stored crop, heads which appear perfectly sound may be badly rotted in the interior. Field losses of 40 to 50 per cent have not been uncommon and losses amounting to 90 or almost 100 per cent have been recorded. A quarter of the stored crop of cabbage was lost at Racine, Wis., in the winter of 1896–1897 (Russell).

Etiology.—This disease is caused by a specific bacterial organism which was first described as *Bacillus campestris* by Pammel in 1895. He proved the pathogenicity of the organism for rutabagas and yellow turnips, by pure culture inoculations and reisolations. In 1896–1897, Smith confirmed the work of Pammel on rutabagas and turnips, using pure cultures from turnips and cabbages, and also made successful inoculations on cabbages, cauliflower, kale, rape, radish and black mustard. Cross-inoculations showed that the disease on the various hosts was caused by the same organism. In the early work of Smith the pathogene was referred to as *Pseudomonas campestris* (Pam.) E. F. S., and this name

was quite generally used by American and European writers until Smith discarded Migula's classification and substituted *Bacterium campestre* (Pam.) E. F. S. In the classification of bacteria proposed by the committee of the Society of American Bacteriologists the organism is described as *Phytomonas campestre* (Pam.) S. A. B.

The rods are 0.4 to 0.5 × 0.7 to 3 microns, and occur singly or in long chains. When crowded in the plant or in old cultures the rods are very short, almost coccus-like, but in young infections or cultures, they are much longer than broad. Young bacteria are actively motile by means of a single polar flagellum. No endospores are formed. The colonies on agar are pale yellow, circular, thin, flat, entire, with slight tendency to form concentric rings (for detailed cultural characters, see Smith, 1911).

The infections of either young or old plants occur largely through parts above ground, but especially through the leaves. Harding reported infections through the injured roots, but Smith has found but little evidence of this type of infection except possibly in seedlings. Infections take place readily through mechanical injuries, and various leaf-eating insects are important agents of dissemination. Aphids and mollusks (slugs) have been shown to transmit the disease. While wounds may be the avenue of entrance of the pathogene, the majority of infections occur independent of mechanical injuries, normal leaf openings—the water pores—being easily entered. These modified stomata occur in groups on the serratures of the leaf margin, and during cool nights exude liquid moisture, which collects in dew-like drops. When weather conditions are such that the drops persist for some time, and the pathogene reaches this moisture, it multiplies and the rods soon penetrate the substomatal chambers, after which the entrance into the adjacent vascular elements of the veins is only a matter of time. The characteristic leaf symptoms with blackening of the veins will follow with some rapidity after the initial incubation period of 7 to 20 days. Smith reports as many as 400 distinct marginal leaf infections on a single plant. The occurrence of infection through ordinary stomata has been denied, but this has recently been demonstrated by Drechsler for the cotyledons of the cabbage, which are devoid of hydathodes. The formation of numerous lesions in the leaf-spot type of disease in cauliflower and in Chinese cabbages would also suggest the probability of stomatal invasion.

The bacteria multiply in the spiral and other vessels, move gradually forward as they increase in numbers, finally reach the base of the leaf and enter the vessels of the main axis. From this point they may be distributed to other leaves, the movement outward or upward being aided by the transpiration stream and the mobility of the rods. After the organism has penetrated the central axis of the host, wilting of leaves may result from the plugging of the vessels in the petioles by bacterial aggregates, even before a general penetration has taken place.

The source of primary infections in any occurrence of the disease may be from contaminated soil or from bacteria which are carried on the surface of the seed. Manure from diseased refuse may contain the pathogene, and in such material or in ordinary soil which has produced a diseased crop it may persist from one season to another. Smith records a serious outbreak of the disease in cabbage in a field that was manured with "refuse from a storehouse in which brown-rotted cabbage had been wintered over." An infected seed bed may cause numerous infected seedlings which will carry the disease to the field. Contaminated seed

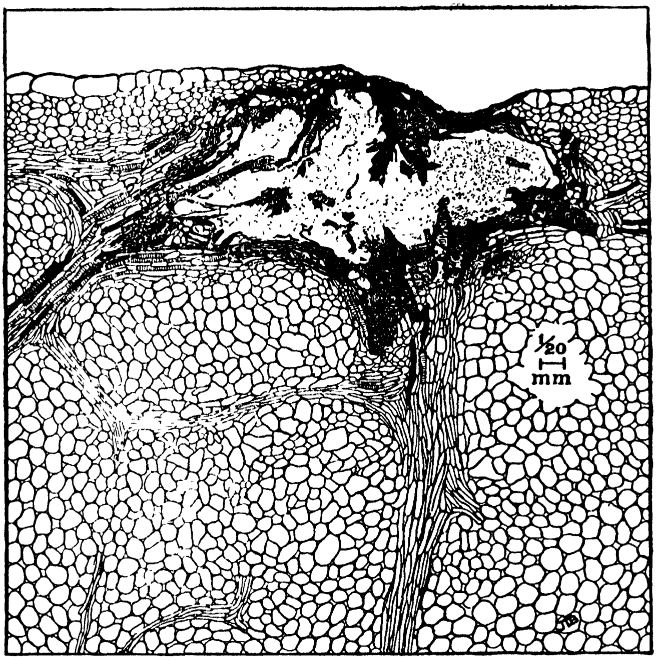

FIG. 92.—Section of a cabbage leaf parallel to the surface near the margin, showing the result of infection by black-rot bacteria through the water pores. (*After E. F. Smith.*)

as a source of infection was first emphasized as an important feature, but further evidence has been offered by Walker and Tisdale, who cite a number of cases in which the disease was introduced into Wisconsin fields by seed imported from Europe. After the disease is established in a field it may be spread by natural agencies from the infected plants to healthy plants. In the case of cabbage and some other crucifers the seed is contaminated during the threshing operation, but in the cauliflower there is a direct infection through the seed pods, so that the bacteria are within the seed coats. This behavior is the probable explanation for the ineffectiveness of the mercuric chloride treatment of cauliflower seed.

Pathological Anatomy.—While first entrance of the bacteria is through parenchyma, the parasite soon penetrates the vascular elements, where it makes its principal development. The spiral and reticulated vessels are very often filled with dense masses of bacteria, and these may sometimes break through into the surrounding parenchyma. "Often the intercellular spaces are first occupied; the middle lamella is then dissolved and the elements are separated and squeezed into all sorts of shapes by the multiplication of the bacteria" (Smith). The lignified elements are not digested; at least the spiral threads and other lignified portions of vessels persist. Non-lignified elements may be entered by the bacteria and destroyed by this means as well as by the wedging apart by the mass action of intercellular development.

The destruction of parenchyma tissue adjacent to bundles in the way described is the cause of pronounced cavities in succulent structures. The advance of the bacteria in the parenchyma is relatively slow, and the cavities rarely if ever reach the surface of the organs in which they are formed. The formation of cavities is common in such structures as cabbage or cauliflower petioles and in turnip roots.

Host Relations.—Practically all of the cultivated species of Brassica are susceptible to the black rot. In 1898 Russell stated that: "In all probability there is but very little difference in susceptibility, all varieties yielding to the disease, if the causal organism is once present." The disease is known to infect cabbage, cauliflower, brussels sprouts, rape, collards, kale, rutabagas, turnips, radishes and mustard. It has been found on winter stock (*Matthiola incana*) in Germany and recently on Chinese cabbage (*Brassica chinensis*), the loose-headed varieties being less susceptible than those with compact heads. Radishes are rather resistant, turnips and rutabagas more susceptible and cauliflower very susceptible. The Houser cabbage has been reported as "practically immune to black rot under field conditions," and is also listed as one of the older domestic varieties resistant to wilt or yellows. It should be noted, however, that the yellows-resistant varieties developed in Wisconsin have not proved resistant to black rot "or to the other common cabbage diseases such as blackleg (Phoma) and club root (Plasmodiophora)."

Prevention or Control.—No single practice will control black rot, but emphasis should be placed on (1) seed disinfection; (2) soil disinfection of the seed bed; and (3) on cultural or sanitary practices in general. If black rot is the only seed-borne trouble to be prevented, the mercuric chloride treatment is satisfactory, but if blackleg is present or suspected, the hot-water treatment should be used. Treatment of seed with mercuric chloride 1–1000 for 30 minutes followed by a cold-water rinse and drying of the seed has been recommended, but it has not proved uniformly effective and is not recommended for cauliflower. Since the

mercuric chloride is not effective against blackleg or for black rot of cauliflower, the hot-water treatment is to be recommended. On the basis of the most recent studies by Clayton, soak the seed in hot water as follows: cabbage and brussels sprouts, 25 minutes at 122°F.; cauliflower, 18 minutes at 122°F. Remove to cold water, drain and spread out to dry. In either treatment the seed can be handled to best advantage in a thin cheesecloth sack. The hot-water treatment causes more injury than the mercuric chloride, so whenever attempted, a preliminary treatment and germination test of a small lot of the seed should be carried out. Uspulun is reported to give excellent results but it has not been sufficiently tested.

Since infections may originate from organisms present in the soil of the seed-bed, some method of soil sterilization should be practiced. It is worthy of note that the treatment of the soil with 1 to 1000 or 1 to 1200 mercuric chloride, which gives good results in the control of the root maggot, is also excellent for black rot. Good control of the disease in cauliflower has been obtained by applying the solution three times to plants in the seed bed. The same treatment is also of value in the control of club root and damping-off due to Rhizoctonia.

Late planting is recommended, especially for cauliflower.

Black rot is a warm-weather disease and can be avoided by planting late enough so that the crop develops during the cool fall months. Generally speaking, fields planted after the last of July are fairly free from black rot (Clayton).

The following additional recommendations have been listed: (*a*) Use manure and soil for the seed bed known to be free from the disease; (*b*) practice crop rotation, using one in which no cultivated crucifers or cruciferous weeds are allowed to grow for 4 to 5 years; (*c*) give attention to the control of all insect pests; (*d*) do not allow live stock to roam from diseased to healthy fields; (*e*) pull and destroy diseased plants as soon as detected, but do not throw them on the manure heap. It should be noted that the practice of removing affected leaves from cabbage or cauliflower and destroying them, which was at one time recommended, has been shown to be without value.

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FIRE BLIGHT

Bacillus amylovorus (Burr.) Trev.

Fire blight is a serious bacterial disease affecting apples, pears, quinces and other species related to the pome fruits, while it also affects some of the stone fruits as a minor trouble. In its various phases it attacks the blossoms, causing a *blossom blight;* the leaves, by direct invasion, producing *leaf blight;* the young growing twigs, causing their death and that of the foliage which they bear, thus producing a characteristic *twig blight;* the older branches or the main trunk of the tree, producing *cankers, body blight,* or if the lesions are located at the base of the trunk, one type of *collar blight* or rot; the young or developing fruit, causing either *fruit spot* or *fruit blight.* Because of the frequency and severity of the trouble on the pear, the disease has been frequently spoken of as *pear blight,* but *fire blight* seems a more appropriate name, since it is suggested by the most common phase of the disease, the twig blight, of the various hosts.

History.—Fire blight is apparently a disease indigenous to North America, where it probably occurred on wild hosts previous to the introduction of the cultivated varieties. It was first observed by William Denning about 1780, in the Hudson River Highlands, but was not described until 1794. From that time on the disease attracted more and more attention as it spread, with the increased cultivation of pears and apples, to the westward and the southward. In the oldest book on fruit culture in America, "Cultivation of Tree Fruits," published by William Coxe in 1817, fire blight was discussed as an important feature of fruit production. While the disease was at first confined to the regions east of the Alleghany Mountains, it worked its way westward and by 1876 to 1880 it had become very destructive in Illinois and other adjacent states of the northern Mississippi Valley. It also spread to the south and completely devastated certain pear-growing districts on the gulf coast of Texas. The regions west of the Rocky Mountains were free from the disease for a time, but it reached California about 1900 and between that date and 1910 had wrought havoc in the pear orchards of that state. It spread later into the newer orchard regions of the Pacific Northwest, being very severe in the Rogue River Valley of Oregon by 1908, while by 1914–1915 it was epiphytotic in the Yakima Valley of central Washington.

Despite the severity of the disease and frequent discussions as to its cause and means of overcoming it, little progress was made between 1817 and 1878–1880, when Burrill discovered and proved its bacterial origin. It is interesting to note the many and varied theories, advanced before Burrill's discovery, to explain the origin of fire blight: (1) electricity or atmospheric influence, since it was especially noticeable after a thunderstorm; (2) the action of the sun in moist, hot weather, the "sun-scald" theory; (3) the "frozen-sap theory," which accounted for the disease by the cutting off of the supply of moisture to the branches; (4) old age, or a phenomenon of senility; (5) overnourishment of trees by high culture; (6) deficiency of the soil in certain essential mineral matter; (7) insects, for example, the ambrosia beetles; (8) fungi; (9) some morbid influence transmitted through the air from tree to tree.

In 1878, Prof. T. J. Burrill, then Professor of Botany in the University of Illinois, first advanced the theory that the blight was due to bacteria which he found to be constantly present in the blighted tissues. His theory was soon substantiated by producing the disease by inoculations of healthy parts with the juice from diseased tissue (1881). His work was completely confirmed by more detailed experiments by Arthur (1885). Since that time numerous contributions have increased our knowledge of the disease and a voluminous literature has developed. It is only possible to mention a few of the landmarks that are outstanding features of our progress: (1) the determination of the dissemination of blossom blight by flies and wasps, by Waite (1898), followed by studies by other workers which have shown the part played by various insects in the spread of blight; (2) the general adoption of surgical methods of control, with the disinfection of instruments and cut surfaces (Waite, Whetzel and others) with 1–1000 mercuric chloride or with formalin (Jones, 1909); (3) study of cankers and body blight of the apple, and their experimental production by inoculations (Whetzel, 1909); (4) a special study of holdover blight (Sackett, 1911); (5) the determination of the intercellular migration of bacteria in the tissues (Bachmann, 1913; Nixon, 1927); (6) special investigation of fire blight as a disease of nursery stock (Stewart, 1913); (7) a determination of the part played by fire-blight bacteria in the production of collar blight or crown rot (Orton, 1915); (8) the recognition of primary leaf invasions of the fire-blight bacteria, thus producing a leaf blight independent of infections through the twigs (Heald, 1915, 1927; Miller, 1929; Tullis, 1929); (9) the establishment of the inefficiency of the mercuric chloride method of disinfection of wounds in cutting-out operations (Reimer, 1918); and (10) the realization of the value of blight-resistant stocks and the inauguration of systematic work in testing of all known pear varieties for resistance, coupled with breeding for resistance.

Geographic Distribution.—The disease occurs throughout the North American continent wherever apples or pears are grown, with the exception of a very few favored localities. From its region of origin in the east it spread westward with the increased cultivation of its two common hosts until it reached the Pacific Coast. It apparently reached Oregon and British Columbia about the same time, but for some reason has failed to develop in Washington west of the Cascades. Sufficient time has elapsed since its arrival in the coast country for it to have become prevalent in western Washington, hence it seems possible that climatic factors are responsible for its absence from this section. For a long time fire blight was confined to the North American continent. It was first reported in Japan in 1911. In 1917 a bacterial blight of pear blossoms was reported from South Africa, but this was shown to be distinct from the fire blight of America and the causal organism was described as a new species, *Bacterium nectarophilum* Doidge. The true fire blight reached far-away New Zealand in 1919, and spread rapidly throughout the North Island, being reported as a serious disease of apple, pear, quince and medlar, and widespread on hawthorn hedges, which are frequently used for windbreaks and fences. A severe epidemic of fire blight on pears was reported from Italy in 1924 (Montemartini, 1925).

Blight upon pears has been most severe in the warmer and more humid regions of the eastern United States, being a limiting factor in the production of some of the best commercial varieties, especially before the introduction of control measures. It has been less severe in the northern range of commercial pear growing, especially around the Great Lakes or in the vicinity of other large bodies of water.

Symptoms and Effects.—The blossoms, leaves, twigs, fruit, limbs or trunk may be directly affected by localized infections, but it is rare for all phases of the disease to be found during the course of a single season in the same environment.

FIG. 93.—Blossom blight of Jonathan apple.

Fire blight may cause a complete blighting of blossoms, or only partial blighting may result. Soon after the blossoms open they may begin to turn brown and wilt and this discoloration soon involves the young fruits. The discoloration then extends down the pedicels, and the adjacent leaves are also involved, turn brown, wilt and remain hanging as a blighted tuft around the blasted group of flowers. The discoloration may then advance down the fruit spur, resulting in the death of this structure. In many of the clusters of blighted blossoms minute, pearl-like or amber-colored droplets will appear on the pedicels. These are likely to appear if the atmosphere is relatively moist, and they may remain as visible indications of the malady or they may be dissipated by

rains. In many cases no further progress will be made, but in others, especially in susceptible varieties, small stem lesions may develop at the base of certain of the blighted spurs, while in others the advance is not checked until larger areas or entire branches are invaded. In certain seasons *blossom blight* is the principal phase of the disease that is evident, and it may appear in epiphytotic form, while in other cases little or no blossom blight may be in evidence.

A direct invasion of leaves may result in leaf blight, which appears in quite characteristic fashion. For many years direct attacks of the leaves were denied, but in 1915 they were shown to be common in various orchard sections of Washington on both apples and pears. The leaf

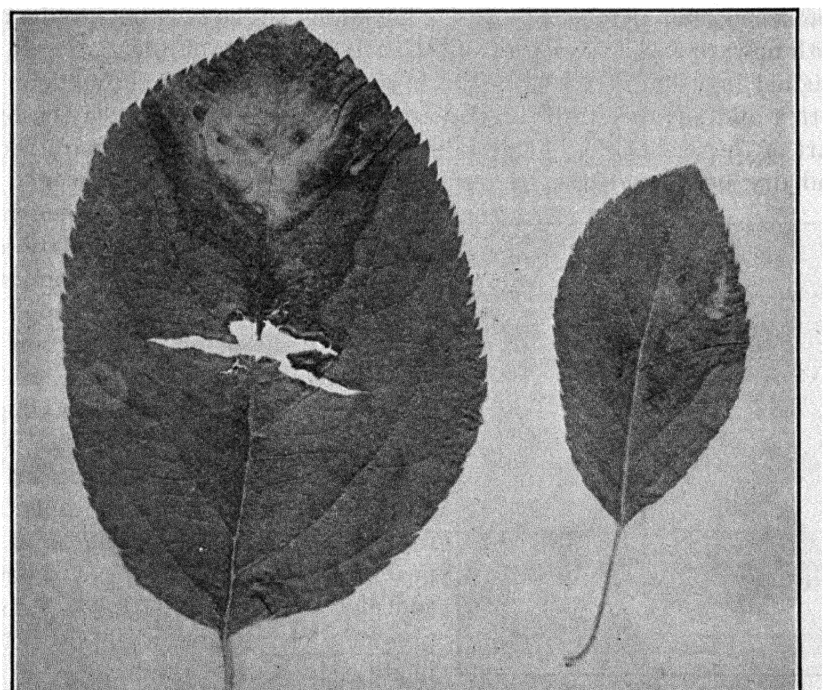

FIG. 94.—Apple leaves showing natural leaf invasions by the fire-blight bacteria.

lesions are generally marginal, being either lateral or terminal, but occasionally they may be at any point on the leaf blade. Under field conditions all gradations of leaf invasions may be observed, from those which involve only small marginal areas to others which have advanced throughout the extent of the leaf blade. In many of the lesions the advance seems to be checked and is never resumed, while in others there is a steady advance until the entire leaf blade is killed and the disease may then spread down the leaf stalk and later result in the blighting of the twig. Marginal leaf lesions are generally roughly triangular, with the point of the triangle following either the midrib or one of the main

lateral veins. The invaded tissues do not show a uniform coloration, but are more or less zonate, from varying shades of brown, always much darker in the pear than in the apple. Bacterial exudate of the same nature as the "pearls" on the blighted-blossom pedicels may ooze out from the leaf lesions on the lower surface or spread out as thin films which dry to form shiny flakes.

Twig blight may result from primary infections in the stem of succulent shoots or from leaf infections and the gradual advance of the bacteria into the twig bearing the leaves. Frequently the first evidence of twig blight in the apple is a faint amber-yellow or reddish coloration of the tip, which begins to wilt and droop, but in the pear the blighting tips are generally blackened, even in relatively young infections. The twig blight advances both upward and downward from the original center of infection. In the original invasion of the twig the bacteria may migrate throughout the cortex and advance into the petioles of leaves and even reach the basal part of the leaf blade, but probably most of the leaves wilt, turn brown and dry out before the bacteria have had time to penetrate into the mesophyll, since the stem lesions have produced a physiological isolation of the leaves to within their limits or distal to them. Viscid drops of bacterial exudate may appear on the main axis, petioles or bases of leaf blades just as in the blossom blight. The drooping terminal twigs with shriveled and curled drooping leaves, either brown, as in the apple and most other hosts, or black, as in the pear, which stand out in marked contrast to the normal green foliage, present a characteristic picture of twig blight. It is this resemblance of the foliage to that blasted by frost or heat that has suggested the name of fire blight. In sudden blighting of twigs during hot, dry weather, the affected foliage may show a much paler color than when average conditions prevail.

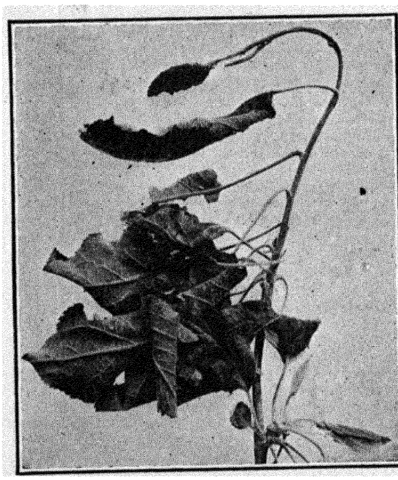
FIG. 95.—Typical twig blight of Jonathan apple.

The disease may advance downward through the twig and reach other branches, through which it may spread, or its progress may be checked at any point. The extent to which the disease has advanced in a twig beyond the appearance of the blighted leaves may be determined by the watery or discolored appearance of the stem cortex. Stem cankers may result from twig invasions, in the same way as described for the blighting of fruit spurs, or the disease may continue to advance into larger limbs or

even into the main body of the tree, killing the invaded structures with the progress of the disease.

While the very young developing fruits may be blasted as a direct result of blossom blight, a *fruit spot or blight* may result from new infections on the partially grown fruits. Infection of pears is much more common than apples, and in pear orchards infested with fire blight the blackened half-grown fruits covered with numerous drops of bacterial exudate are a frequent spectacle. Pear fruits are so susceptible to fire blight that, if once infected, the disease is not checked until the entire

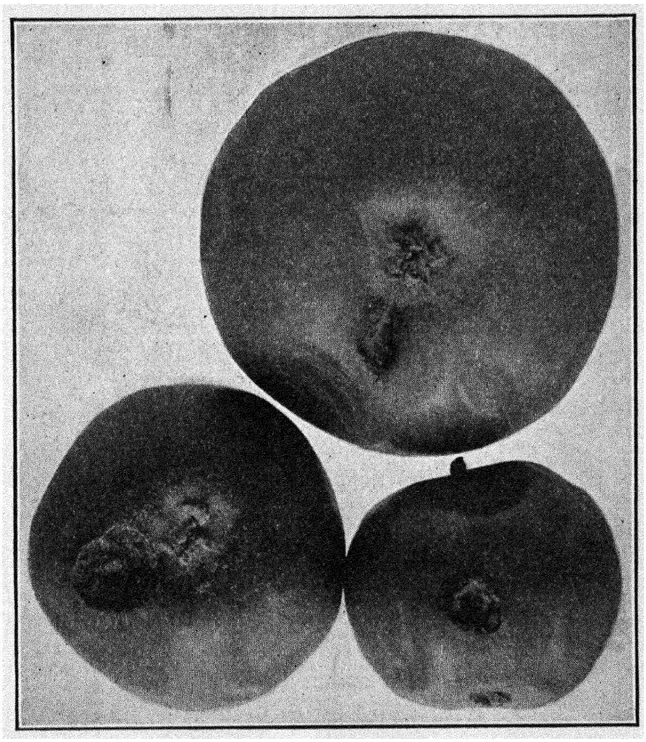

FIG. 96.—Fire-blight lesions on green apples.

fruit has been invaded. This behavior is also true for some of the more susceptible apple varieties, but in the more resistant species or when infections occur in fruits well advanced in development, circular or slightly irregular lesions may result, which cease to advance. In these the invaded tissue shrinks and turns dark brown or almost black, the bacteria die out just as in twig infections and the lesion remains as a sunken discolored area.

When the disease invades limbs or main trunks of a susceptible host, cankers of limited size may result (*blight cankers*), or the disease may continue to spread slowly or rapidly, according to conditions, throughout the entire season, and produce a *body or limb blight*. The production of

definite cankers is common in apple varieties and a very high percentage of these originate from primary infections in fruit spurs, normal leafy twigs or water sprouts. In active cankers the bark is slightly darker than normal and appears as if water soaked. It may blister and rupture, and characteristic gummy exudate or bacterial ooze may be forced out, the amount depending upon the size of the canker and the succulence of the tissues invaded. In large cankers the ooze may be produced in such quantity as to run down the side of the tree trunk or limb. When

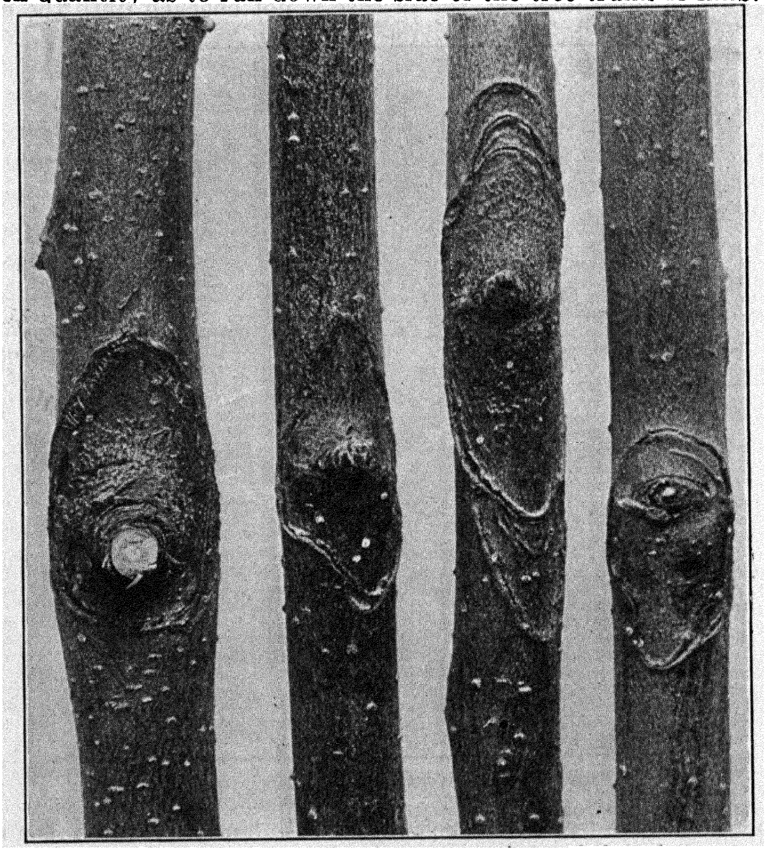

FIG. 97.—Fire-blight cankers on young active apple branches.

cut into, the diseased bark will appear brown and water soaked, in marked contrast to the pale green or creamy white of normal bark. The bacteria may advance rather uniformly in all directions through the bark or may make more rapid extension along a rather narrow pathway. In the apple the majority of cankers generally reach their maximum size rather early in the season and the bacteria, unable to make further advance, die out either from lack of nourishment or because of other unfavorable conditions. When a lesion ceases to spread, the tissue dries out and shrinks somewhat and a split or crack appears, separating the old dead tissue

from the surrounding normal bark. The bacteria may remain alive at some point in a few of the well-defined cankers, and resume activity the next spring when growth starts and thus cause the extension of the canker. Such cankers are called *holdover cankers*, and serve frequently as centers from which the disease may be spread to adjacent healthy trees. In the pear, especially on susceptible varieties, limited cankers are much less frequent, and when limbs or the main trunk are infected, the disease is likely to spread until the entire tree is killed, unless its advance is checked by surgical methods.

In active fire-blight lesions it is an easy matter to demonstrate the presence of the bacteria by placing a few fragments of the brown, sappy tissue in a drop of water. In a few moments the bacteria begin to swarm out of the affected tissue and cause a turbidity or milkiness of the water, which is quite evident and characteristic, especially when viewed over a black background. In cankers which are old or in which activity has ceased, the bacteria have largely disappeared, so that general symptomology must usually be relied upon for diagnosis. The bark of old cankers may be invaded by saprophytic fungi; and in many localities *Cytospora* is a common accompaniment, the yellow spore horns or tendrils appearing during damp weather.

When blight lesions develop near the base of the tree trunk close to the ground level, the form of the disease known as *collar blight* results. Trees affected by this form of the disease will show an unhealthy appearance of the foliage, especially on certain limbs. The noticeable features are reduced size and number of leaves and more or less pallor or yellowing. There are no sudden wilting and browning of the foliage as in twig blight, but premature defoliation may result. The examination of trees showing this symptomology will show a lesion of some extent at the crown, a region of dead, discolored and sunken bark. This lesion advances rapidly in the early part of the growing season, but generally is arrested by midsummer. Activity may be resumed the next season, and the lesion may spread until the trunk is girdled at the base, which leads to the death of the tree. Under other conditions, the blight may spread rapidly upwards through the trunk and into some of the branches, causing sudden death before basal girdling has been completed. The diseased tissue at the crown appears brown and water soaked, the same as in other blight lesions, and a distinct line of demarcation may separate it from the normal bark. In old lesions the bark may separate from the sapwood, which first becomes brown or later almost black.

Collar blight may be mistaken for collar rot due to winter injury or for some form of fungous root rot. Winter-injury lesions usually show on the southwest side of the trunk, are practically full size early in the spring and so show no progressive advance during the growing season. The water-soaked appearance of the bark so characteristic of collar

blight is lacking in collar rot due to winter injury. Secondary invasions of fungi may result through either collar blight or lesions of collar rot produced by severe winter conditions.

The injury from blight varies with the location, severity or number of lesions. As a result of the disease the following injuries may result: (1) loss of foliage; (2) blighting of blossoms, and consequently the failure to set fruit; (3) spotting or blighting of fruit that has escaped the ravages at blossoming time; (4) the dieback of twigs and branches from twig blight or limb cankers which girdle the axis on which they form; (5) lowered vitality and poor growth of partially affected branches from body blight or collar blight that fails completely to girdle the affected parts; (6) death of entire tree from body blight or from collar blight that girdles the trunk near the ground level. The injury may be slight, the crop may be ruined by blossom blight and the tree survive with little other damage or the affected tree may be disfigured, crippled and doomed to make a struggle for existence or it may succumb outright.

Etiology.—Fire blight in its various phases is an infectious and contagious bacterial disease due to the specific pathogene, *Bacillus amylovorus* (Burr.) Trev. Burrill first established the bacterial nature of fire blight and named the associated organism *Micrococcus amylovorus* (1878–1881) because of the incorrect belief that it was a destroyer of starch, but the binomial was changed by Trevisan to the one now in general use. Stewart's discovery that the specific name first published by Burrill was *amylivorus*, and therefore should be used, is of no importance, since Dr. Burrill reported that this spelling was a typographic error. In the "Manual of Determinative Bacteriology" arranged by the Committee of the Society of American Bacteriologists, the organism appears under the name of *Erwinia amylovora*. The first cultural studies of the organism, using various media, were made by Arthur in 1887. Inoculations were made with bacterial ooze and with pure cultures, with the production of the characteristic symptoms of the disease, and numerous workers have attained similar results since that time. Previous to 1915 all of the different phases of the disease except leaf blight had been recognized, but workers had failed to secure infections through the leaves. The writer studied fire blight in Washington in the summer of 1915 and found leaf invasions common in pear, apple, and quince, and later (1927) reported artificial infections through the leaf margins by the use of pure broth or bouillon cultures. Leaf infections occurred through marginal breaks, insect punctures or through perfectly sound leaves. Brooks (1926) was unable to secure stomatal infections, but Miller (1929) obtained stomatal infections through foliage leaves, sepals and receptacles, while Rosen (1929) reports similar results, with entrance also through the stomata of peduncles and even infection through petals which bear no stomata.

B. amylovorus is a short rod form averaging about 0.6 by 1.5μ with maximum size of 0.9μ in diameter and 1.8μ long. The shortest forms (1μ) appear almost spherical or oval, but they are really short cylinders with rounded ends. The cells are mostly single, often in twos, and sometimes in threes or short chains, 2-4 flagellate, the flagella generally being located at or near the poles, and no endospores are known. The claim of Rosen (1926) that the rods have a single-polar flagellum appears to have been based on a faulty technic, as Bryan (1927) has pointed out. The life-cycle idea of Nixon (1926, 1927) in which the bacteria change form, with progress of the infection with the culmination of overwintering cysts, has not been verified by other workers. The organism grows well on the various culture media. (For further details see work of D. H. Jones (1909) and also that of V. B. Stewart (1913).)

The blight bacteria are not able to survive the winter in the soil or in the dead parts of their host. Holdover cankers are the centers from which blight starts in the spring. Bacterial ooze from such cankers may be washed down by rains or carried away by insects, and if cankers are active at the time of blossoming, the blossom blight may result. Only a small percentage of the cankers produced will persist as holdover cankers capable of yielding living bacteria and starting an extension of the lesion in the spring. Sackett (1911) found that 25 per cent of 83 pear cankers examined by him contained living bacteria; Brooks (1926) found, 2.5 to 11 per cent of active cankers and 0.6 to 2.5 per cent of blighted twigs with living bacteria persisting through the winter. There appears to be great variability in the percentage of holdover blight under varying conditions. Holdover cankers on any host are a source of danger, as the bacteria may be transmitted from one host to another without any loss of virulence. The disease may thus be carried from pear to apple, apple to pear, or from quinces or hawthorns used for windbreaks or as ornamental plantings to any commercial orchard of apples or pears. It has recently been claimed (Rosen, 1930) that the beehive may be the first source of blossom-blight infections, even before any hold-over cankers have begun to ooze. In support of this observation he reports the recovering of the blight bacteria from beehive material in summer, winter and in the early spring prior to the development of blight.

The first authentic evidence as to the dissemination of the fire-blight bacteria was contributed by Waite (1898). He showed that flies and bees visit oozing cankers and carry away the bacteria on their legs and mouth parts, and plant them in the nectar of open blossoms in their visits from flower to flower. These bacteria find the nectar an excellent food and multiply rapidly in it and finally invade the blossoms through the nectaries, thus producing the characteristic blossom blight. Continued visits of insects to the inoculated blossoms may still further spread the

disease, and after centers are established, especially if they are near the top of a tree, a large amount of new infection may result from contaminated rain water (50 to 90 per cent, according to Gossard and Walton). The bacterial exudate of leaf and twig blight may also be washed down by rains and play a part in producing new infections. Other studies have shown in more recent years that insects play a very important part in the transmission of the blight bacteria and in the production of wounds through which they gain entrance into leaves, twigs and branches. Certain species of aphids or plant lice, several leaf hoppers, the tarnished plant bug and some bark-boring beetles have been shown to be carriers. It is only reasonable to believe that almost any sucking insect, or any with a bark-burrowing habit, may be a carrier, if sources of bacteria are present and the insect is one that feeds upon a susceptible host. Specific data are available to show that heavy infestation of an orchard with aphids, for example, increases the amount of fire blight in case the disease is present. It has also been shown that birds, like the sapsucker, may be agents in the transmission of the disease (Waite). Man himself may also be the agent by which the disease is carried, since it has been shown in numerous cases that the blight bacteria may be carried on the surface of pruning tools which come in contact with blight lesions. Cases are also on record in which blight lesions appeared in large numbers by infections through wounds caused by hail, the mechanical injuries serving as the avenues of entrance into which the bacteria were washed by the rains. A claim has recently been made that the bacteria of fire blight are also wind borne (Stevens *et al.*), but the evidence submitted does not justify such a conclusion, except in connection with wind-blown rain. Recent workers seem to attach increased importance to the part played by meteoric water in the dissemination of the blight bacteria from the primary centers of infection and less to the migrations of insects.

It has been shown that the bacteria of fire blight may remain viable and virulent after being kept two months in distilled water and that they were able to live in dried exudate for a period of 9 months. Such facts suggest that possibly hold-over cankers and the beehive are not the only means by which the bacteria are carried over from one period of infection to another.

Pathological Anatomy.—Diseased tissues show a necrosis or death of cells, the first effects being plasmolysis, digestion of middle lamellæ and later digestion of cell walls and penetration. Rosen claims that the cells are surrounded by bacteria and killed by asphyxiation. The bacteria may be found in great numbers in the intercellular spaces of the invaded tissue, but opinions differ as to the exact condition in which they exist. Early reports (Bachmann, 1913) pictured them as floating free in intercellular liquid, and Miller later presents the same view for young infections but states that the later condition does not represent a true zoöglœa,

although the matrix is more viscous than at first. Nixon (1927) describes them in the initial stages of advance as embedded in a jelly-like zoöglœa, which he believes migrates with pseudopod-like extensions, pushing the cells apart by pressure. Whatever the method, there seems to be an agreement that cells of invaded tissue may be separated, sometimes with the production of cavities (schizogenous) or pockets which become filled with the bacteria. In the later stages of penetration when cells are invaded and digested, the cavities may be larger and of lysigenous origin. The first and principal migration of the bacteria is in the parenchyma tissue of the parts invaded, leaves or stems. In stems the cortical tissue is penetrated first, with deeper tissue later, the bacteria finally reaching phloëm, cambium, wood parenchyma and pith in the more extreme cases, but the degree and extent of penetration are variable and may be checked at any stage. The bacteria may even break into fibrovascular bundles in leaves or stems. The cortical spread of the pathogene is the basis of the successful application of scarification and of the use of the zinc chloride treatment without cutting. During periods of pronounced activity the bacteria accumulate in such quantities that they are forced to the surface either through natural openings or fissures or ruptures and flow out as the characteristic bacterial exudate, as described under Symptoms. It does not seem to be quite clear what part is played by enzymes, osmotic pressure, surface tensions and mass-mechanical action in separating the cells and producing the ruptures which permit the bacterial ooze to reach the surface. Nixon (1926) explains overwintering of the pathogene by the formation of what he terms cysts or pseudo-fructifications, that is, cells filled with the bacteria in dense aggregates on the outer edges of cankers. The existence of such cysts is denied by Miller (1929) and by Rosen (1929).

Predisposing Factors.—It may be stated as a general principle that any conditions which favor a more succulent and rapid type of growth render a host more susceptible to the ravages of fire blight. Under such conditions infections are more likely to occur and the advance or spread of the lesions will be more rapid and will continue longer without being checked. This is in accord with the recognized fact that the young growing tissues in the first part of the growing season are more susceptible than the older tissues which have ceased activity and assumed their mature condition. The prevalence of blight is influenced by weather conditions, cultivation practices, the use of irrigation water and the application of fertilizers. Prolonged hot and dry weather is unfavorable to blight, retarding dissemination and increasing resistance of the tissue, while abundant rainfall followed by warm, cloudy weather is favorable (65 to 85°F. with relative humidity of 80 per cent or more), since dissemination is more likely, while the tissues become gorged with cell sap and are more easily invaded. In some sections blossom blight will not occur

unless there are active blight cankers producing exudate which can be disseminated when the blossoms are in the susceptible state. This is not necessary in Arkansas according to Rosen (1930), who reports beehives as the sources of the pathogene for first infections. Pears, especially in the Pacific Northwest, generally escape blossom blight, because they generally come into flower before holdover cankers have become active, but exudate is being produced in time to start an epidemic of blossom blight in apples. Clean cultivation promotes fire blight largely through stimulation of growth and the production of succulent tissues, in contrast to orchards in sod or some cover crop. Fire blight is likely to be severe in irrigated orchards, since the use of irrigation water is frequently excessive and promotes a more rapid growth than occurs in many non-irrigated localities. Fertile soils supplied with abundant moisture favor fire blight in contrast to poorer soils which lead to a slower and less succulent type of growth. Stewart (1921) demonstrated that twig blight is increased in severity by heavy applications of stable manure or of other nitrogenous fertilizers, and Orton (1915) makes a similar claim for the collar-blight type of the disease.

Host Relations.—Fire blight has been of first importance as a disease of pears, many varieties of which are exceedingly susceptible to the trouble. It is of next importance in the apple orchard. While it occurs sometimes upon apricots, plums, prunes and cherries, it is a disease of minor importance on these hosts. The principal phase of the disease on prunes is the twig blight of either nursery stock or young trees. A fruit blight of cherries in the Yakima Valley was shown to be due to the blight organism (Hotson, 1915). The quince is very susceptible to fire blight, and while of little importance as a fruit it frequently serves as the producer of holdover cankers, which are a menace to the commercial plantings of apples or pears. As ornamental shrubs or trees, quinces are frequently neglected and allowed to produce holdover cankers, unmolested. Fire blight also occurs on the loquat (*Eriobotrya*) in Florida, Georgia, Texas, and California and has recently been reported (1921) as affecting the medlar (*Mespilus*) in New Zealand. The disease also attacks certain other wild or cultivated species, as wild crabs (*Malus*), hawthorns (*Cratægus spp.*), service berry (*Amelanchier canadensis*), the red-berried California holly (*Heteromeles arbutifolia*), the American mountain ash (*Sorbus americana*), and the European mountain ash (*S. aucuparia*), Japanese flowering quince (*Chænomeles lagenaria*), the fire thorn (*Pyracantha coccinea*), wild and cultivated strawberry (*Fragaria spp.*), raspberry and blackberry (*Rubus spp.*), rose (*Rosa spp.*), and spiræa (*Spiræa vanhoüttei*). The English hawthorn which is used in ornamental plantings or for hedges is very susceptible, and during the blight epidemic of 1914–1916 in Washington it was frequently found to be severely affected. The rather general plantings of hawthorn hedges for windbreaks around

orchards in New Zealand has seriously complicated the control of fire blight in that country, since they served as a harbor for holdover blight to be carried into adjacent fruit trees.

Practically all of our cultivated pears and apples are very susceptible to blight when growing in the nursery under conditions favorable for the development and dissemination of the malady. Opinions vary somewhat, however, as to the susceptibility of the different varieties under orchard conditions. Of pears the Bartlett, Howell and Flemish Beauty are generally reported to blight severely and rapidly, while the Kiefer, Seckel and Winter Nelis are more resistant. Blight is sometimes checked in grafted trees when the disease in the susceptible scion reaches the more resistant stock. The search for resistant pear stocks suitable for commercial use has recently occupied the attention of agricultural investigators. Certain Asiatic species and especially *Pyrus calleryana* and certain forms of *P. ussuriensis*, brought from China by F. C. Reimer, are of especial promise. The need for a resistant or immune stock is especially urgent to prevent the serious losses from body and collar blight.

There is a general agreement that the Transcendent crab and the Esopus Spitzenberg are extremely susceptible. It is not uncommon to find the Transcendent crab very heavily blighted, while surrounding trees of other varieties are only slightly affected. From such very susceptible trees the disease may spread later to the more resistant varieties if conditions continue favorable for its development. In many localities Spitz trees are being discarded because of their extreme susceptibility to blight. Reports on the resistance or susceptibility of various other apple varieties vary so much for the different localities that their behavior cannot be predicted with certainty.

Preventive or Control Measures.—Ever since fire blight has been known, unscientific and unreliable methods for its cure or control have been proposed from time to time. Even in recent years unsubstantiated claims have been made of the effectiveness of internal dosage or of external applications—patent nostrums or blight specifics. These remedies has been put on the market either by unscrupulous manufacturers or by well-meaning but misguided enthusiasts. Suffice it to say that none has stood the test of science and practice. When fire blight once becomes established in a region, eradication is out of the question, and its severity may be expected to vary from season to season, sometimes smouldering, sometimes bursting out into epiphytotic form. The palliative or control practices which are available fall mainly into five categories: (1) modification of the susceptibility of the host; (2) control or elimination of insect carriers; (3) the avoidance of very susceptible hosts as windbreaks or for ornamental plantings in the vicinity of orchards; (4) tree surgery; (5) the use of resistant or immune varieties or stocks.

1. Attention should be given to planting and to cultural and irrigation practices and an attempt should be made to produce a normal growth with no excessive stimulation and overrapid growth. Trees which have made a moderate but firm, thrifty growth will be in better condition to withstand blight when it does strike. In case the disease appears in severe form or becomes epiphytotic it will be advisable either to discontinue or to modify those practices which promote the formation of rapid-growing, succulent shoots. These practices must be varied to meet the needs of different environments—sometimes the use of less irrigation water or the cessation of cultivation, the development of a weed cover or of a seeded cover crop to utilize the surplus moisture, the avoidance of barnyard manure or other nitrogenous fertilizers, the avoidance of heavy or excessive pruning, or permitting the trees to produce a heavy crop with only moderate thinning. Pruning trees so as to produce an open or vase type of growth, with as great freedom as possible from water sprouts and fruit spurs near the main trunk or on the large branches, will lessen the danger of body blight or collar blight.

2. The recognized importance of certain insects in the transmission of blight certainly dictates that known carriers should be checked in their development in so far as possible. Merrill (1915) has cited a striking illustration in the case of aphid control: Jonathan trees sprayed with Black-Leaf 40 were practically free from blight, while adjacent unsprayed trees were very seriously infected. Selby (1915) has emphasized the importance of using contact insecticides, stomach poisons and repellant sprays and barriers to give the best possible control of the insect carriers, such as

> . . . plant lice, red bugs, the tarnished plant bug, bees at blossoming time (?), bark beetles, burrowing into twigs, and probably many others which bite the leaves or buds, suck the sap from the twigs, make incisions into the bark to lay their eggs or creep into the crevices and wounds for feeding and shelter.

McCown (1929) reports that a 1–3–50 Bordeaux applied when trees were in full bloom reduced the infection 52 to 79 per cent for apples and 91 per cent for pears.

3. In New Zealand the experience with hawthorn hedges and windbreaks has shown the folly of using a very susceptible host for such a purpose in the planting and planning of commercial orchards. The most remote corner of the world may be reached by fire blight sooner or later, so the menace of susceptible hosts as possible bearers of holdover cankers should always be kept in mind. The neglected orchard must also be recognized as a menace.

4. The surgical treatment for blight should begin during the period of winter pruning. Careful inspection should be made and all cankers, especially those on the trunk or larger limbs should be cut out in order to

lessen the number of holdover cankers. If all blight lesions could be cut out, the disease could be eradicated, but the most careful examination fails to locate every case. Early spring examinations, if made carefully and frequently, should reveal cankers that have been overlooked and these may be removed before exudate has been formed or before much opportunity has been offered for dissemination. Small branches bearing lesions should be removed by a cut several inches (4 to 6) below the external evidence of diseased tissue. In case of cankers on large limbs or trunk, the diseased tissue should be completely removed, and the cut extended well into the adjacent healthy bark. None of the diseased prunings and material excised from cankers should be allowed to lie around in the orchard, but should be collected and destroyed at once. In cutting out blight, the cutting instruments and cut surfaces should be carefully sterilized. When the surgical method was first put into operation, mercuric chloride or corrosive sublimate, 1 to 1000, was adopted as the standard sterilizing agent and for many years its effectiveness was not questioned. Reimer has shown that it is ineffective for sterilizing cut surfaces, since the mercury combines with the organic substances from the cut tissue to form albuminate of mercury, an inert compound. It is, however, effective in sterilizing steel instruments. Mercuric cyanide has been found effective on cut surfaces but not on steel instruments, so a combination of the two is now being recommended: 1 ounce of each to 4 gallons of water. After disinfection, the exposed surfaces made in cutting out lesions or pruning large limbs should be protected by a coating of either Bordeaux paint or asphalt paint. This coating should be renewed from time to time so as to keep the wounds thoroughly protected. In cases of trunk cankers or collar blight with lesions that extend over halfway around, surgical treatment will be of doubtful value unless bridge grafting is resorted to.

More recently (Day, 1924) a modification of Reimer's formula has been proposed as follows:

Mercuric chloride		1 ounce
Mercuric cyanide		1 ounce
Glycerine	3 parts	3½ to 4 gallons
Water	1 part	

This has been shown to be more effective than the old formula consisting of the mercuric cyanide and corrosive sublimate alone. The glycerine increases the penetration of the disinfectant and delays drying so that the chemicals remain active for a longer time. The addition of fuchsine red or other water-soluble aniline stain has been recommended[1] to give color to the treated surfaces so that the operator can be sure that all cut surfaces have been covered.

[1] CARDINAL, H. A.: *Science* **60**: 455. 1924.

The scarification method is recommended (Day, 1924) for the treatment of infections on the trunk or large limbs, but this should be carried out before the blight has penetrated too deep. In this treatment the dead outer bark is shaved down with a knife or special scraper until most of the diseased tissue is removed. Special attention should be given to the crotches, and the thick bark there and elsewhere should be cut away nearly to the cambium, but not too deep, as the cambium may be injured by the penetration of the disinfectant. No surface bark should be left anywhere in the scarified area and the cutting should extend 4 or 5 inches beyond the advancing edges of the canker. As soon as scarification is completed the entire cut surface is painted with the mercury-glycerine disinfectant. It is reported that careful workmen in California secure control in 80 to 95 per cent of the cankers treated by this method. All hopelessly diseased trees should be cut out and destroyed at once, as they are a constant menace to the other healthy trees. In treating collar blight the soil should be removed from around the crown and all diseased tissue cut out, using the same method of sterilization as for other locations. Allow the wounds to dry thoroughly and then coat with asphaltum paint or lead paint (preferably the latter according to Orton), and after this has dried carefully replace the soil around the collar.

Since the scarification method was too slow and laborious to be practical during severe epidemics, a more rapid method has been adopted by some California pear growers (Day, 1928). New cankers on the larger branches or trunk are painted without any cutting with a zinc chloride solution made up in three strengths, 53 per cent, 43 per cent and 33 per cent in water, hydrochloric acid and denatured alcohol, the strengths being varied according to age of trees, size of roots or branches and the temperature which prevails. It is claimed that with this method 85 to 90 per cent of treated cankers are cured, with careful work. It must be emphasized that the treatment is somewhat uncertain, because of danger of killing by too deep penetration of the solutions.

The need of practicing summer cutting of blight will vary with the locality and with the varieties of fruits involved. In the majority of cases summer cutting in apple orchards is not necessary if careful attention is given to the removal of all holdover cankers during the winter or early spring. In the case of pears, especially the more susceptible varieties, summer cutting out of blight may be necessary to hold the disease in check.

5. The selection of resistant varieties offers some relief, but unfortunately many of the most valuable commercial fruits are highly susceptible, especially pears. The hope for a solution of the problem for pear growers lies in the discarding of the susceptible French seedling stocks which have been generally used, and substituting the most valuable resistant stock.

Of all that are available, certain strains of *Pyrus ussuriensis* and *P. calleryana* offer the most promise.

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CROWN GALL AND HAIRY ROOT

Pseudomonas tumefaciens (S. & T.) Duggar and *Pseudomonas rhizogenes* (Riker *et al.*)

These diseases manifest themselves on various woody or herbaceous plants by the formation of tumor-like enlargements at the crown or on other parts or by an excessive production of organs (roots, shoots). In both cases the abnormal development is an uncontrolled hyperplasia, resulting in tumors or galls of varying size and form or in the production of organs, frequently out of normal position and in excessive numbers. Because of the frequent location of the galls on the stem or trunk at or near the ground level the disease has been known as crown gall, but various other names, such as crown knot, root knot, root tumors, cane galls and black knot, have been applied to the tumor forms with slight or no root production, while hairy root, woolly root and woolly knot are names applied to the phases of excessive root production.

History.—Typical crown gall of various plants has been known for more than half a century. Early French and German writers ascribed the trouble to frosts or to mechanical injuries. This disease has been recognized by nurserymen in the United States for many years, and has been the subject of much discussion and study since 1890. It has been suggested that the disease was introduced into this country with European importations since "Wurzelkropf" of apples and pears, which is apparently identical with crown gall, was described by Sorauer as occurring in Germany in 1886 and earlier. Owing to its wide distribution in the United States, crown gall has been a frequent subject for investigation by Experiment Station and Federal workers, and as a result a voluminous literature has developed. Between 1890 and 1900 varying opinions were prevalent in the United States concerning the nature of crown gall, some workers pointing to its probable infectious character, while others decided "that these root galls are not the work of a parasite, but are a malformation following some injury of the root or some uncongenial condition in soil or treatment" (Bailey, 1896). Cavara in Italy (1897) was the first to prove the bacterial nature of the disease on the grape, but his work, as well as that of several other writers of southern Europe, was either overlooked or discredited. In 1900, Toumey published an "inquiry into the cause and nature of crown gall," in which he demonstrated the infectious nature of the disease and arrived at the conclusion that the causal organism was a new species of slime mold to which he gave the name of *Dendrophagus globosus*. The writer recognized the incompleteness of his work and the uncertainty of his conclusions, but no positive proof of his error was forthcoming until Smith and Townsend of the Bureau of Plant Industry (1904–1906) isolated a bacterial pathogene from galls of the Paris daisy, *Chrysanthemum frutescens*, and were able to produce galls readily by inoculations with pure cultures. They gave the name of *Bacterium tumefaciens* to this organism, which has since proved to be the cause of the various hyperplastic responses characteristic of this disease on various hosts. It seems probable that Hedgcock isolated the same organism from grapes in 1903 and again in 1904, since he records successful puncture inoculations from an organism producing a white colony.

The hairy-root disease was first brought to our attention by Stewart, Rolfs and Hall in 1900 by their description of the simple form on apple seedlings. This and three other forms of hairy root have since been recognized by Hedgcock (1908–1910). What is apparently identical with the aerial form of hairy root has been described as "Kropfmaser" by Sorauer (1886) and Frank (1896) and more recently by other European workers.

Since the publication of the comprehensive bulletin by Smith, Brown and Townsend (1911), Smith has published numerous papers showing the relation of crown gall to human cancer. C. O. Smith working in California has contributed valuable data (1912; 1925) concerning the symptoms and effects of the disease on many different hosts. Recent work by Riker (1923) and by Robinson and Walkden (1923) are worthy of note since the conclusions are not in accord with the earlier observations of E. F. Smith.

Since the determination of the bacterial origin of crown gall, workers have been directing their efforts again to find effective control measures and also to find out the degree of injury caused by the disease in the orchard. In this connection mention may be made of the work of Ness in Texas (1917) and of Melhus and Maney in Iowa (1921) on control; of Swingle and Morris in Montana (1918) and of Green and Melhus in Iowa (1919) on extent of orchard injury. The papers and resolutions resulting from the crown-gall symposium held at Cincinnati, Dec. 28 to Jan. 1, 1923–1924, have been printed and distributed by the American Association of Nurserymen (February, 1924) and give valuable data on the amount of injury from the disease. The detailed studies during the past few years by workers at the Wisconsin and Iowa Experiment Stations (Keitt, Melhus and coworkers) have been fostered by some financial support

from the American Association of Nurserymen, and it is the culmination of these investigations that has revealed the etiological separation of crown gall proper and hairy root or woolly knot as distinct diseases.

Geographic Distribution.—Crown gall on various hosts is now known in practically all parts of the world, but, in general, it reaches its greatest severity in the regions of high temperatures, as may be illustrated by the fact that it is more abundant in the southern states from Virginia to Texas than in the more northern states, although it is not uncommon in Canada. In some of the more northern localities the disease seems to be especially severe on certain hosts—for example, on blackberries in the Puget Sound country and on raspberries in Wisconsin. Recent investigations have shown the marked influence of soil temperature and moisture in the development of the disease, so it may be that abundant moisture in certain cases will more than counterbalance the retarding influence of moderate temperatures. Although crown gall has been known in Europe for many years and has been reported from Asia, South Africa, New Zealand and South America, most of our knowledge concerning the disease has come from American workers.

Symptoms.—Two general types of abnormal growth are characteristic of crown gall: (1) typical overgrowths or tumors, *true galls*, of varying

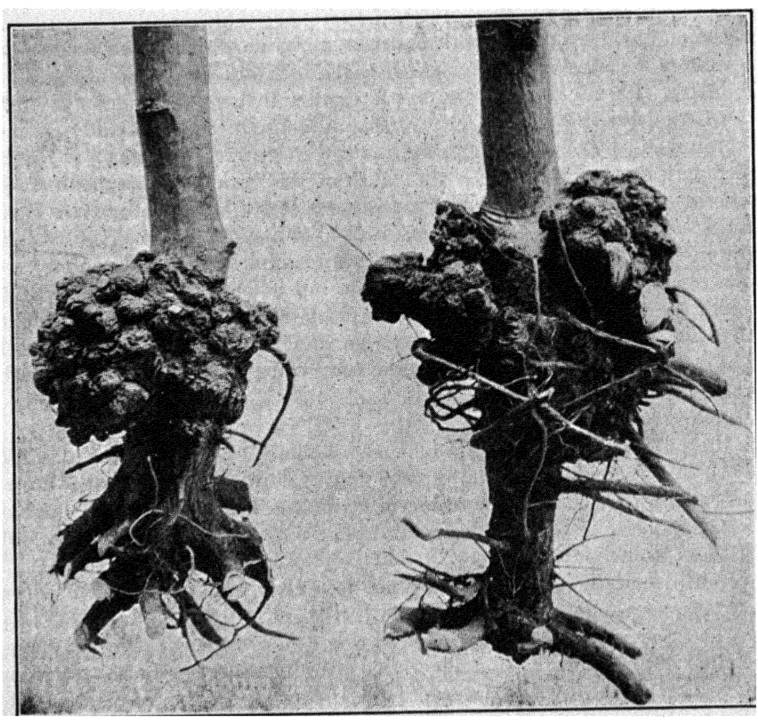

FIG. 98.—Crown gall on young apple trees.

form and size, located on the crowns, roots, stems or leaves; (2) excessive or abnormal development of organs either with or without an accompanying tumefaction. The true galls on fruit trees are so commonly found at the base of the trunk just below the ground level, on the part called the crown, that the common name "crown gall" was suggested, and this

name is frequently applied to the tumors found on other parts of the host. These galls may be irregular globular or elongated in form with more or less convoluted surface, the size varying somewhat with the size and vigor of the structure from which they originate, sometimes exceeding this six to ten times in diameter. In actual size they vary from that of a pea to gigantic overgrowths weighing 50 to 100 pounds, although this maximum size is rather uncommon. The surface of young galls is almost white at first, but this later changes to the color of surrounding normal

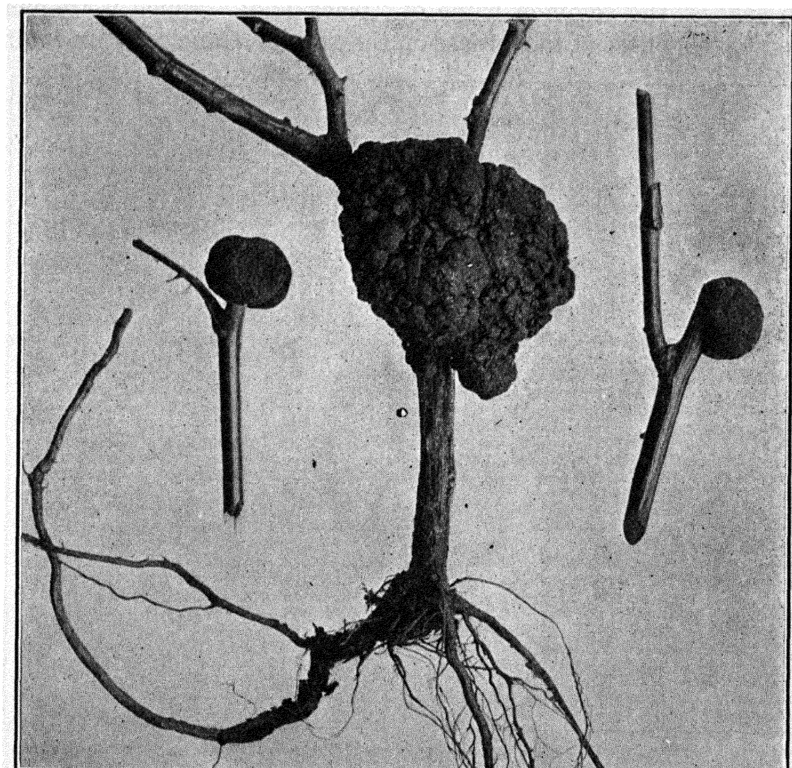

FIG. 99.—Crown gall on roses grown under glass. Infection through pruning cuts.

parts and may even become darker from the decay or weathering of the surface cells. Most of the tumors are made up of succulent and imperfectly vascularized tissue, and have been termed "soft crown galls." These soft crown galls on young woody roots or on herbaceous plants generally decay at the end of the growing season and do not produce roots from their surface. In perennial, woody hosts like the apple, the tumors may persist and develop a covering of bark and an interior woody structure, constituting the so-called "hard crown galls." In the light of recent investigations it seems probable that many of these hard galls are in reality overgrowths or callus enlargements resulting from graft misfits (Riker and Keitt, 1926; Melhus, 1926; Muncie, 1930).

In some hosts, such as the daisy, grape, quince, apple, blackberry, rose, willow and poplar, the galls are very frequently produced on the aerial parts as well as at the crown or on the roots. Cane galls may occur on the grape, often up to 3 to 5 feet above the ground, in the form of isolated or confluent excrescences that are often more or less elongated parallel to the length of the cane. These are at first like wound tissue or callus in appearance, but later in the season become dark brown or almost black due to weathering and decay of the outer tissues, and in this condition suggest the common name of "black knot," which is frequently applied to this phase of the disease on the grape. Cane galls are common

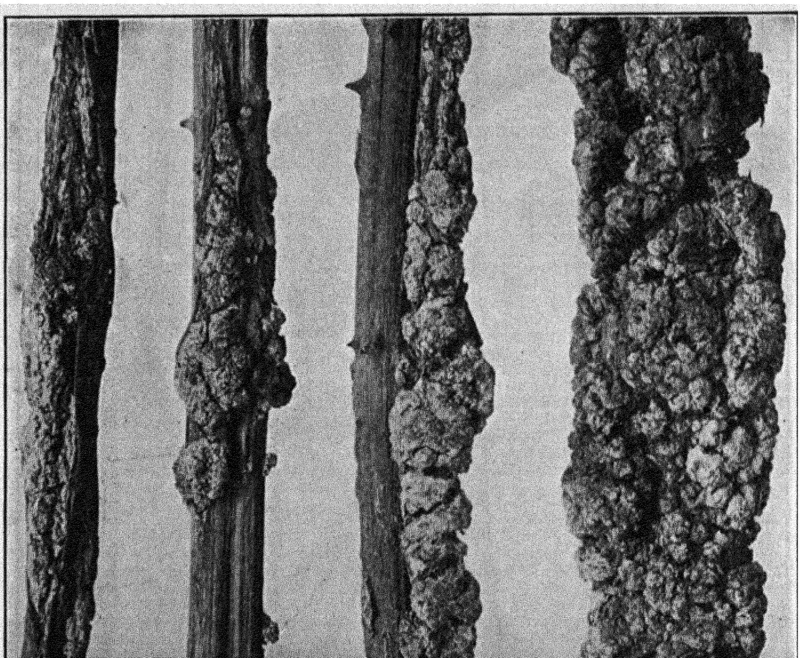

FIG. 100.—Blackberry canes showing different degrees of injury from cane galls.

on the blackberry, especially in the Puget Sound country. These are generally in the form of very much elongated, more or less convoluted excrescences frequently involving the entire circumference of the cane and result in longitudinal splitting and cracking.

In the hairy-root disease there is an increased and abnormal production of roots or of incipient roots, the following forms being recognized: (1) *simple hairy root*, characterized by numerous small wiry roots growing singly or in tufts from the main root or from the base of the stem with little or no enlargement; (2) *the woolly-knot* form, initiated by the formation of a tumor, which soon produces many fine roots from its surface, with intricate branching and frequently with fasciation; (3) the *broom-root* type, consisting of a broom-like grouping of fine roots, usually at the end

of a side root; (4) the *aerial form*, appearing first on trunk and limbs as smooth fleshy swellings developing incipient adventitious roots internally, which, with progress of the malformation, break through the bark and produce warty knots. Under normal conditions these roots make no further development, but cuttings containing the earlier stages of the disease will produce normal roots if supplied with suitable moisture conditions or if placed in moist soil. The aerial form is macroscopically indistinguishable from burrknot (Swingle, 1925).

Recent observations and investigations (E. F. Smith) have shown that aerial tumors may give rise to leafy shoots or flower buds as well as roots, the type of development depending upon the kind of tissues infected. Various types of these artificial teratomas are figured by Smith ("Introduction to Bacterial Diseases") on cauliflower, castor-oil beans, tobacco, Pelargonium and oranges. For example, an infection on the cut end of an internode of a tobacco stem produced 100 closely bunched, leafy shoots. Artificial infections of various hosts have also resulted in the formation of typical fasciations, and the same writer has recorded a typical rosette or witches'-broom development of the carnation which was proved to be due to the crown-gall organism. The extent to which fasciations and similar abnormal developments which occur in nature may be manifestations of the crown-gall organism or of the hairy-root organism is uncertain, but the experimental results have shown that bacteria are at least one of the possible causal factors in these diseases.

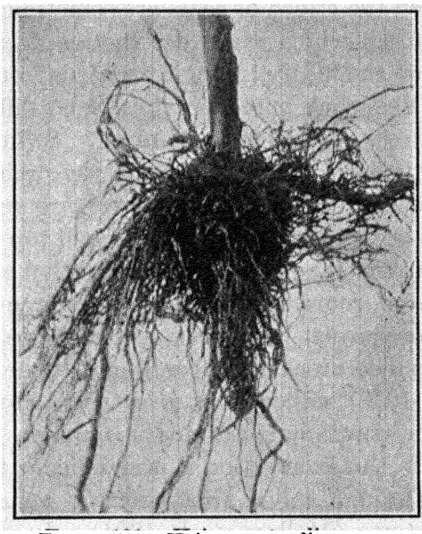

FIG. 101.—Hairy-root disease on grafted apple trees. (*After Hedgcock, U. S. Dept. Agr. Bul.* **90**, *Part II.*)

We may have all varieties of stimulating secondary effects on normal tissue from prolapsis of uninjured leaf and flower buds and root anlage located in the vicinity of tumors, through simple fusions or divisions (fasciations), to the breaking up of the dormant bud, or of a cambium into dozens and even hundreds of small vegetative fragments which may either grow as roots or shoots on the surface of a tumor or be buried in its depths (E. F. Smith).

Economic Importance.—Crown gall and hairy root manifest themselves in such a variety of ways on so many different hosts that it is impossible to generalize as to the kind and extent of injury which may result. The amount of injury varies with the host, its age at time of infection,

location of the tumors, number of infections and the type of host response. In the various cases the following effects have been recorded: retarded growth and dwarf development with undersized, chlorotic foliage; the killing of branches, canes or roots from the presence of localized cancerous lesions; and more rarely the death of the entire plant. In fruit trees the infections of young stock are more likely to cause injury than infections on older trees. The various writers agree that crown gall is a serious and destructive disease of almonds, peaches, plums, grapes, blackberries and raspberries, and is directly or indirectly responsible for the unprofitable condition of plantings in various sections of the country. The lesions on the crown or roots are sometimes avenues of entrance of rot-producing fungi which cause destructive decay. The reports of different workers on the injury to apple orchards from crown gall are somewhat conflicting. Hedgcock noted practically no difference between healthy and diseased trees 6 years after planting. Swingle and Morris (1918) report observations on affected trees in the orchard through a period of 7 years and conclude that affected trees are generally poorer than those unaffected, the disease causing dwarfing of top and especially of the root system, with interference with food conduction and mechanical weakening of the crown and roots. According to their evidence, "it would be better for an orchardist to pay the regular price of healthy stock than to plant crown-galled stock if he could get it free." According to Greene and Melhus (1919), galled trees grow more slowly than normal ones, with reduction in trunk diameter and in number, length, thickness and weight of twigs. In this connection it is significant to know that the rate of flow of water through galled apple stems is reduced by 30 per cent over that of normal stems (Melhus *et al.*, 1924). In general, hairy root seems to be of less consequence than the typical tumors. In the light of recent work it is uncertain whether true crown gall was always used in these tests.

The losses are not confined to the orchard, since there is frequently a heavy infection of nursery stock at the time it is dug for delivery. The present practices in nursery inspection call for the rejection and destruction of all stock showing any evidences of the disease, regardless of the species or variety involved. Nursery losses up to 70 per cent have been recorded. Recent work, however (Riker and Keitt, 1925, and later workers), has led to the conclusion that a large per cent of the malformations called crown gall or hairy root are merely the results of excess callus formations.

Etiology.—True crown gall is caused by a specific bacterial pathogene, *Pseudomonas tumefaciens* (S. & T.) Duggar. The organism was first isolated from galls on the Paris daisy by Smith and Townsend (1904–1906) and described as *Bacterium tumefaciens,* while according to the classification of the Society of American Bacteriologists it is *Phytomonas tumefaciens.* The pathogenicity of this organism was demonstrated by numerous

successful inoculations on various hosts, and it has since been repeatedly isolated from true crown galls on various hosts by other workers. Using the strains from the daisy and hop, E. F. Smith recorded the production of galls on 40 kinds of plants belonging to 18 families. C. O. Smith using a strain isolated from the peach made successful inoculations on 30 or more species and varieties of fruit, nut, and shade trees. Following the work of Smith and his associates there was a tendency to attribute various aerial and subterranean overgrowths or malformations to the crown-gall organism, frequently without adequate proof. It has been shown by more recent studies that many of the overgrowths on apple-nursery trees discarded in nursery inspection as crown gall are in reality the result of graft misfits (Riker and Keitt, 1925, 1926; Melhus, 1926; Muncie, 1930). As an illustration, Riker and Keitt found that 86 per cent of 407 crown-gall rejects of apple-nursery stock did not yield any crown-gall bacteria when tested by cultures.

At first it was the belief that all forms of crown gall including hairy root were due to the single species or strains of that species, but several different workers have shown that there are marked differences between the apple organisms causing woolly knot and hairy root and the ones that cause typical crown gall on various hosts (Siegler, 1928, 1929, 1930; Muncie, 1930). This differentiation of the two forms (Riker *et al.*, 1930) has culminated in the recognition of the hairy-root organism from nursery-apple trees as a new species, *Pseudomonas rhizogenes*. These workers isolated the hairy-root bacteria from 78 of 96 enlargements of the hairy-root type including woolly knot and reproduced typical hairy root by inoculation, followed by the recovery of the organism. Also the hairy-root organism inoculated on apple stems above ground gave malformations which resembled small burrknots. Similar results should be credited to Siegler, who previously (1928) reported the production of malformations identical with the aerial form of hairy root (burrknot) by inoculation of apple with the woolly-knot strain. It appears to be true that hairy root and burrknots (Brown, 1924; Swingle, 1925) may arise independent of the stimulation by *P. rhizogenes*, but the evidence appears to be conclusive that a certain percentage at least of these malformations are of bacterial origin. Swingle (1925) has contended that many of the knots described as aerial hairy root are in reality burrknots or normal root developments peculiar to certain varieties. Whether the failure to isolate the hairy-root bacteria from burrknots, as reported by several workers, indicates their non-parasitic origin is an open question, since there may be an early disappearance of the pathogene, as others contend.

The following are some of the more important differences between the two organisms, both of which are typically small rods producing small, slow-growing, circular colonies that are raised and glistening translucent (Riker *et al.*, 1930):

Crown-gall organism: Not motile in hanging-drop culture; shows strong absorption of Congo red; size 0.75 to 2.25 by 0.30 to 1.05μ, average 1.43 by 0.60μ; produces smooth galls devoid of roots.

Hairy-root organism: Motile in hanging-drop culture; shows weak absorption of Congo red; size 0.55 to 2.59 by 0.15 to 0.75μ, average 1.44 by 0.43μ; produces galls with roots.

Muncie (1930) reports at least two strains of hairy-root bacteria, one of which has polar flagella and is closely related to *P. tumefaciens*, while the other is stated to have peritrichous flagella. There seem to be some discrepancies in the observations on flagellation by various investigators. Hill *et al.* (1930) claim that crown-gall isolations are motile in hanging drop but not when taken direct from the host.

The crown-gall bacteria are intercellular, occur in large numbers and appear to be largely confined to the more superficial portions of the gall (Robinson and Walkden, 1923) rather than intracellular and few in number, as was at first reported by E. F. Smith. It has been demonstrated that the bacteria advance in the intercellular spaces in the form of zoöglœal strands (Robinson and Walkden, 1923; Hill *et al.*, 1930), and it is contended that this advance may account for secondary tumors rather than the invasive growth of tumor tissue or tumor strands, as had been contended by Smith.

The crown-gall and hairy-root bacteria are very widely distributed organisms and are apparently native in many soils, where they may lead an independent life or persist in old galls. It has been shown that crown-gall bacteria can overwinter in the soil under Iowa conditions at a minimum temperature of −32°C. They were kept in sterilized soil in the laboratory for 736 days (Patel, 1929); also infection of tomatoes was successful from both sealed and unsealed soil cultures after over 500 days.

Both of the groups of organisms appear to be unable to enter normal, uninjured tissue, but they can readily establish themselves through mechanical injuries of many types, such as pruning and grafting wounds, insect injuries, etc. The presence of the bacteria does not kill the affected tissue but stimulates the cells and thus causes an abnormal and rapid cell division, some cells being reduced in size, and others enlarged, leading to the formation of tumors or to the abnormal development of organs. The period of incubation before the appearance of an evident gall varies from 5 days to several weeks on various hosts, and in some cases the organism may remain dormant for months before any evidence of an infection can be noted. The gall formation is dependent upon the growth of the host.

The presence of the bacteria on the surface of developing galls will explain a number of features: (1) the difficulty of isolating the causal organism from the interior of galls; (2) the continued meristematic activity of the tissues close to the surface; (3) the ease with which soil may

become contaminated, since water must wash many of the surface organisms into the soil. The bacteria may reach wounds directly from contaminated soil in many ways: by irrigation waters or splashing and washing from rains, by contaminated pruning tools, by insect carriers or possibly by wind-borne soil. The entrance through grafting wounds will explain the frequent position of large tumors at the crown of the apple and other fruit trees.

The development of crown gall is influenced by the temperature, soil moisture and soil composition. Galls were readily formed on tomato at 18 to 22°C.; they were small and poorly developed at 29 to 30°C.; and at 36 to 38°C. none was formed. In carefully regulated air chambers no galls were formed above 30°C., although the tomato plants grew fairly well. The largest galls for all percentages of moisture were produced at 22°C. and were largest at 60 per cent moisture for all temperatures up to 30°C. (Riker, 1926). *B. tumefaciens* is intolerant of acid conditions of the soil. The limiting point in bouillon is given as pH 5.70 by Smith and Quirk (1926). The pH in the meristematic tissues of tumors on tomato and Paris daisy is given as 5.2.

Brief mention may be made of the claim (Lieske, 1928) that the crown-gall organism is a polymorphic form passing through a definite life cycle of the following stages: (1) filterable-virus stage; (2) typical *Pseudomonas tumefaciens;* (3) a gram-positive form isolated from tumors; and (4) a Streptococcus form. Rosen (1926) figured budding forms and claimed the recovery of typical crown-gall forms from the filtrate passing through a Berkefeld V filter. These reports are in keeping with the recent claims of life cycles for other bacteria.

The comparisons of crown gall to malignant tumors in man has resulted in the application of the name "plant cancer" to crown-gall hypertrophies. Homologies have not been substantiated, and the general consensus of investigators at present is that *P. tumefaciens* will not cause animal cancer, although there are some conflicting studies. Kaufman has recently reported 200 inoculations with various strains on animals with negative results.

Host Relations.—Crown gall has been found to occur naturally on the pome fruits, all of the stone fruits, various species of Rubus (blackberries, raspberries and loganberries), currants and gooseberries, grape varieties, nut trees, such as walnuts, pecans and almonds, numerous woody and herbaceous ornamentals, several deciduous-leaved trees, alfalfa, cotton, beet, turnip, salsify, parsnip and hop. In addition to the natural infection, many herbaceous and woody hosts have been successfully infected with pure cultures by E. F. Smith, C. O. Smith and others. Inoculations made by C. O. Smith on the following gave negative results: "loquat; silk oak (*Grevilliea robusta*); German prune; *Prunus ilicifolia* (wild hill cherry); *Anona cherimolia;* avocado; olive." Natural infections and

artificial inoculations have shown a wide range of susceptibility among the different species and in some cases in the different varieties of the same species. Observations and experimental tests have failed to demonstrate any appreciable increase in resistance of a host as a result of repeated infections. In a comparative study of 45 varieties of Prunus by C. O. Smith (1917) the per cent of infection ranged from 0 to 100. *P. pumila*, *P. ilicifolia* and *P. caroliniana* remained free from infections. Some of the more resistant varieties were: Italian prune (*P. domestica*), 7 per cent; German prune, 10 per cent; Damson (*P. insititia*), 10 per cent; as contrasted with Elberta peach, 94 per cent; Royal apricot, 97 per cent. "Our most popular stocks, as Myrobalan, peach, apricot and almond, are very susceptible, which only goes to confirm field observations that the stock used for the stone fruits is very susceptible to crown gall." The further search for resistant root stocks has resulted in the discovery of promising resistant varieties of both Prunus and Amydalus (C. O. Smith, 1925). The cherry seems to be more resistant than other stone fruits, the Mazzard root showing more resistance than the Mahaleb. English walnut roots are much more susceptible than those of the California black walnut (*Juglans californica*) which is now used as a root stock. The aerial form, called black knot, is very common on quinces in the southern states, California and the Pacific Northwest, but it does not seem to cause serious injury to this host (possibly some of these are non-infectious burrknots). Grape varieties showing resistance are: Concord, Catawba, Delaware and other American varieties, while the European varieties, such as Muscat, Mission, Malaga and Flame Tokay, are more susceptible. Nurserymen report Ben Davis, Early Harvest, Yellow Transparent, Wealthy, Grimes, Northern Spy, Oldenburg, Wolf River, Red June, Gano and Rome Beauty apples as susceptible, but comparisons show variation in resistance of the same varieties from different regions. Greene and Melhus report the Wealthy much more susceptible than the Jonathan.

Since the definite separation of crown-gall and hairy root is a recent development, there may be some discrepancies in the earlier reports as to resistance. Recent studies have shown the tomato and tobacco practically immune to hairy root (Siegler, 1930). Inoculations with the hairy-root organism have been successful on apple, rose, honeysuckle, sugar beet, bean and Paris daisy (Riker *et al.*, 1930).

The basis for resistance to the crown-gall organism is believed to be the acidity of the cell sap (Smith and Quirk, 1926). They attribute the immunity of *Begonia lucerna* to the high acidity of the cell sap and report that various other immune plants have cell sap more acid than pH 5.70. Muncie (1930) reports crown gall on *Rumex crispus* and on rhubarb, both of which are strongly acid. Infection in such cases is by mass action, and the bacteria adjust the reaction ahead of their advance.

Prevention and Control.—This is complicated by the large number of susceptible hosts grown under a great variety of conditions, by the possibility of cross-inoculation from the various hosts, by the widespread occurrence of the causal organism in the soil and by the abundance of the disease in nursery stock. The practices that have been recommended may be discussed under the following:

1. *The Use of Disease-free Stock.*—This is provided for in part by nursery inspection, but the grower should always carefully examine young stock before setting in the orchard and discard any that shows evidences of the disease. Non-infectious overgrowths should also be discarded. In replanting small fruits, like raspberries or blackberries, plantations showing the disease should be avoided as a source of stock. It is also recommended that trees should be examined in the orchard at the end of the first year and either removed or treated if found infected.

2. *Dipping of Planting Stock.*—Since there are many opportunities for apparently disease-free stock to be carrying the disease, treatment of planting stock with a fungicide has been recommended by Ness (1917):

> The bundle of trees should be plunged deep enough into the solution so that the trees are covered to a distance of several inches above the collars of the roots, using 7 ounces copper sulphate to 26 gallons of water for peaches and 1 pound to 26 gallons for apples, with an exposure of 1 hour.

3. *Care in Making Grafts and Their Protection or Sterilization.*—This is of prime importance, since most infections of nursery stock occur through the grafting wounds. Hedgcock obtained good results by wrapping root grafts with cloth and nearly as good protection by "a continuous-thread wrapping applied by a machine evenly and closely over all the union." Packing in sand and cold storage are recommended for root grafts that must be kept for some time before planting. Melhus and Maney (1921) state that "a cloth wrap at present is impractical from an economical standpoint in the production of apple grafts," and present evidence to show the value of dipping in an 8-8-50 Bordeaux, either with or without lead arsenate. Some promising results have also been obtained by planting in soil to which inoculated sulphur had been added at the rate of 600 pounds per acre (Sherbakoff, 1925). Waite and Siegler (1928) have recommended dipping seedlings and scions before and after grafting in Semesan (1–400). Oppenheimer (1926) did not find this method effective but did get results when the grafts were puddled in a mixture of the fungicide, soil, and water. The Semesan treatment did not prevent galls on tomato (Riker and Keitt, 1926). Since non-parasitic overgrowths are more common than crown gall and are also objectionable, they should be reduced to the lowest possible minimum by care in grafting, the most important feature of which is the exact fitting of stock and scion.

4. *The Prevention of Wounding.*—This applies to young stock in the nursery and also to young trees in the orchard. Breaking the bark with

the hoe or other cultivating tools at the crown is a common source of danger.

5. *Rotation or the Selection of Clean Ground.*—This is of especial importance since clean stock may become infected by planting in contaminated soils. This is of more importance in the susceptible stone fruits than in the more resistant apples. Land from which an infected planting has been removed had better be planted to some immune or at least highly resistant crop before replanting to stone fruits. It is stated that newly cleared land may sometimes be contaminated with crown gall which occurred on the native trees or shrubs.

6. *Surgery.*—Badly infected, young trees should be removed and destroyed rather than treated. Cutting out of the tumors on young or old trees is unsatisfactory, since the galls may develop again at the edge of the cut, but some good may result. If galls are removed the work should be carefully done and the cut surface sterilized by coating with a Bordeaux paint (bluestone, 1½ pounds + 1 gallon water; lime, 3 gallons + 1 gallon water; mix equal parts for use).

7. *Sanitary Practices.*—Avoid mixing diseased and healthy nursery stock at digging time. Separate out the diseased stock and destroy by burning as soon as possible. Remember that the bacteria are easily washed off from the galls and may contaminate the soil or infect any susceptible stock. When planting a new tree in the place of an old diseased tree, either use fresh uncontaminated earth for filling in around crown and roots or sterilize the soil with the standard formaldehyde drench (1 pint to 6 gallons, ½ gallon per square foot) and aerate before planting. Follow the general plan of destroying all material showing crown-gall malformations. Neglect to do this may lead to a severe epiphytotic, for example, of aerial galls on roses under glass.

8. *Use of Resistant Varieties or Resistant Stocks.*—Consideration of resistance should govern choice of varieties when consistent with the horticultural needs of an environment (see Host Relations). Probably most attention has been given to the study of resistant stocks adapted to the stone fruits.

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CORKY SCAB OR ACTINOMYCOSIS OF THE POTATO

Actinomyces scabies (Thax.) Güssow and other species

This widespread disease of the potato is characterized by the formation of raised, level or depressed, scab-like areas upon the surface of the potato tuber. Various common names have been applied to the trouble, such as common scab, potato scab, brown scab, Oöspora scab, American scab, deep scab, but corky scab or actinomycosis of the potato have been suggested by Lutman (1914) as the most appropriate names. It should be noted that the potato suffers from several other troubles to which the name "scab" has been applied, for example, powdery scab (*Spongospora subterranea*), black scab or Rhizoctonia scab (*Corticium vagum*) and silver scurf, sometimes called silver scab (*Spondylocladium atrovirens*). The disease in France is called *gale* and in Germany *Schorf*.

History and Geographic Distribution.—The first mention of scab was made in 1825 (Loudens' "Encyclopedia of Agriculture") as follows:

"Scab, that is to say, the ulceration of the surface of the tubers, has never been explained in a satisfactory manner. Some attribute it to the ammonia from the dung of the horse, others to alkali and certain others to the use of wood ashes on the soil. Not using diseased seed and planting in other soil are the only known means of preventing the malady." "Since the organism is believed to be native to the soil, and according to our present knowledge and observation, exists almost universally in soil, especially in those which are well cultivated and rich in humus, the disease is probably as old as is potato culture. Doubtless it has existed both in America and in Europe since first potatoes were grown" (Lutman, 1914).

The early European literature is somewhat confusing because of the occurrence of the powdery scab in that continent, and Humphrey (1889) called attention to the fact that all forms of scab were not alike, and that the "Schorf" of Germany and the common scab of England and America were identical. Various theories were proposed to explain the prevalence of corky scab, the possibility of parasitic origin being suggested

as early as 1842, but it was not until 1890 that claims were made by Bolley that the disease was of bacterial origin and that Thaxter isolated and described the true causal organism. The supposed discovery of the parasitic nature of the disease led Bolley to make the first tests of seed disinfection (1890–1893) with corrosive sublimate, which continued to be the prevailing disinfectant until the introduction of formaldehyde. The use of formaldehyde predominated until it was shown by Gloyer (1913) that it was not so efficient as mercuric chloride in the prevention of Rhizoctonia, a trouble that has a very wide distribution. Important contributions to our knowledge of scab have been made by Lutman and Cunningham (1914), and Lutman (1919) and these writers were the first to recognize the fact that the causal organism described by Thaxter belongs to a group of the higher or filamentous bacteria, the Actinomycetales, although such a suggestion had been made by Krüger in 1905. During recent years the experimental work has been largely directed along two main lines: (1) determination of the effectiveness of the different methods of seed disinfection or soil treatment; and (2) the study of the influence of various environmental factors on the amount of disease.

Symptoms and Effects.—Based on both symptoms and causal organisms several types of scab have been recognized: shallow scab, deep scab and knobby scab (Wollenweber, 1920). In later studies in England six types have been described:

1. *Superficial,* showing merely brownish abrasion of the skin.
2. *Ordinary scab* with irregular concentric layers of cork around a central core or depression.
3. *Pitted scab,* circular to irregular lesions, becoming deep to form a pock or depression.
4. *Stud scab* forming an elevated warty growth (2 to 3 mm.) with abrupt sides.
5. *Tumulus scab,* similar to stud scab but with sloping sides.
6. *Pimple scab* or small, soft, pimple-like pustules (Millard and Burr, 1926).

The scab lesions may show as slightly raised or bulging, roughened, corky areas, or these may be nearly on a level with the remainder of the potato skin, or a corky depression may occur. The lesions may be small and few in number or numerous and larger and produce a general infection with little or no normal surface remaining. Some observations and experiments have shown a tendency for the scab lesions to be segregated on the "stem-end" portion of the tuber, except that this condition is the least evident when potatoes are subjected to the temperatures at or near the optimum for scab development.

The normal symptoms may be modified by the work of wire worms, white grubs, mites or millipeds, which frequently extend and deepen the lesions. There are various other superficial tuber lesions which can generally be recognized by the following characterizations:

1. Black scurf or Rhizoctonia scab, showing as black bodies, "the dirt that will not wash off."
2. Powdery scab (*Spongospora subterranea*), with rather regular, circular lesions, with powdery or granular center.

3. Skin spot (*Oöspora pustulans*), appearing as slightly raised or depressed, dark, circular lesions which do not generally rupture the skin (see account of Powdery Scab).

4. Silver scurf (*Spondylocladium atrovirens*), marked by irregular silvery patches, within which minute dark specks, the causal fungus, are visible.

5. Enlarged lenticels, small groups of raised, whitish cork cells occupying the position of the normal structures.

6. Wart (*Synchytrium endobioticum*), small or large, brown or dark warts which originate at the eyes.

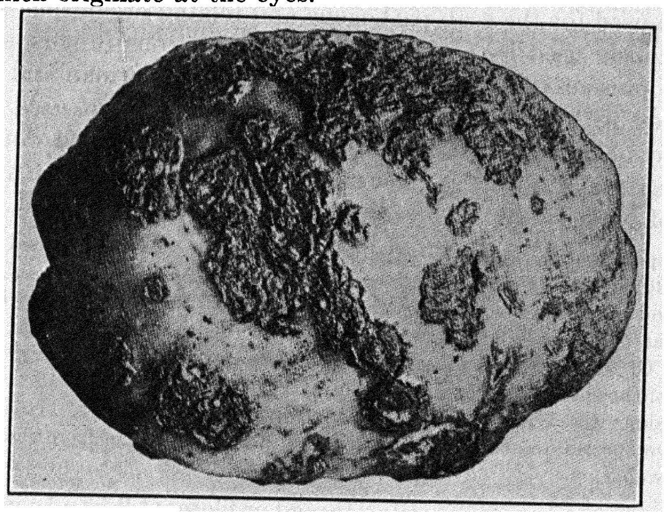

FIG. 102.—Corky scab of potato. (*After Lutman and Cunningham, Vt. Bul. 184.*)

The prominent effects of scab may be enumerated as follows:

1. Lowering of quality. Scabbed potatoes are unsightly; it is necessary to remove a thick paring when preparing the potato for cooking, thus causing a loss; heavily infected tubers have a peculiar earthy odor or taste to which buyers frequently object; there is increased shrinkage in storage owing to greater water loss (Lutman, 1929); and there is increased liability to tuber decay as a result of the penetration of various rot-producing fungi, such as *Fusarium spp.*, Penicillium and Aspergillus.

2. Reduction in yield. When badly scabbed potatoes are used for seed or when the crop is heavily scabbed there may be a very marked reduction in yield. Careful experiments have shown that untreated scabby seed may cause a reduction equal to one-fifth to one-sixth of the crop as contrasted with the same treated seed (Lutman and Cunningham), while Goff recorded a yield of 477¾ pounds from scab-free seed and only 199⅜ pounds from tubers very badly scabbed.

Etiology.—Corky scab of potatoes is caused by various species of Actinomyces, belonging to the Actinomycetales, one of the orders of the

higher or filamentous bacteria. Probably the most common and important species causing scab is *Actinomyces scabies* (Thax.) Güssow. This pathogene was first isolated and proved to be causally related to the disease by Thaxter, who described it as *Oöspora scabies* (1891). His results have been confirmed by various workers since that time, although our ideas as to the botanical relationship of the causal fungus have been changed. The idea that the scab organism belongs to the "higher bacteria" was first expressed for the beet organism by Krüger (1905) and for the potato organism by Cunningham (1911), who placed it in the genus *Streptothrix.* According to Güssow (1914), this generic name cannot be used for the scab pathogene and related organisms, because it was previously used by Corda for a hyphomycetous fungus, so he proposed the present name. Later in the same year Lutman and Cunningham agreed with Güssow as to the generic position but used the name *Actinomyces chromogenus* Gasperini. It has been pointed out that the "Chromogenus group" as recognized by Lutman and Cunningham consists of parasitic (*A. scabies*) and of non-parasitic forms.

Seven species of Actinomyces capable of causing scab were described by Wollenweber (1920). More recently Millard and Burr (1926) studied 24 isolations from potatoes and other sources, one of which was identical with *A. scabies* while 19 were described as new species, 11 of which were able to produce some form of scab on potatoes. Of all the pathogenic species, *A. scabies* was shown to be the most virulent. Virulence of the species was expressed numerically, ranging from 1 to 12. The various types of lesions were connected with infection by particular species. It is not certain whether the species described by Millard and Burr are distinct from those described by Wollenweber.

The organism is a non-motile "thread or filament 0.5 to 1μ in diameter, long and branched, wavy or curved, irregularly segmented and capable of forming aerial hyphæ which break up into gonidia or short cells resembling bacilli." These may form by the "segmentation of any part of the mycelium, either in the depth of the medium or on the surface, giving the colony an ashy gray, cretaceous appearance." These aerial gonidia are not always produced, but may be formed "quite abundantly and in fairly young cultures, while others never produce aerial hyphæ or gonidia, or, if so, rarely and under exceptional conditions" (for detailed cultural characters see *Vt. Bul.* **184**).

Scab lesions originate by the penetration of the pathogene through young lenticels, and the period of susceptibility seems to depend on the degree of suberization of the lenticular tissue.

The organism enters a young lenticel in which subsequently the meristene is stimulated and gives rise to radially elongated cells. Some of these become infected and eventually collapse, and at the same time the collapsed cells become brown tinted, giving the young scab its characteristic color (Jones, 1931).

The area invaded is deepened and broadened and finally cut off by a cork barrier. This cork layer may be penetrated and deeper tissue

invaded with the formation of another cork barrier, the progress probably being checked only by the maturing and harvesting of the tubers. It is supposed that in early stages of infection the parasite produces a pH gradient favorable to cell division, but later conditions favoring suberization are set up, and wound cork results. In the raised form of scab only one wound-cork barrier is formed.

The scab organisms may be present in the soil, or they may be introduced into uncontaminated soil by the use of scabby seed. Later studies have served to emphasize the almost universal occurrence of the scab organism in soil, not only in cultivated land but even in the raw desert land of the western United States. The organism grows and multiplies rapidly in soil rich in humus or containing manure or in the compost

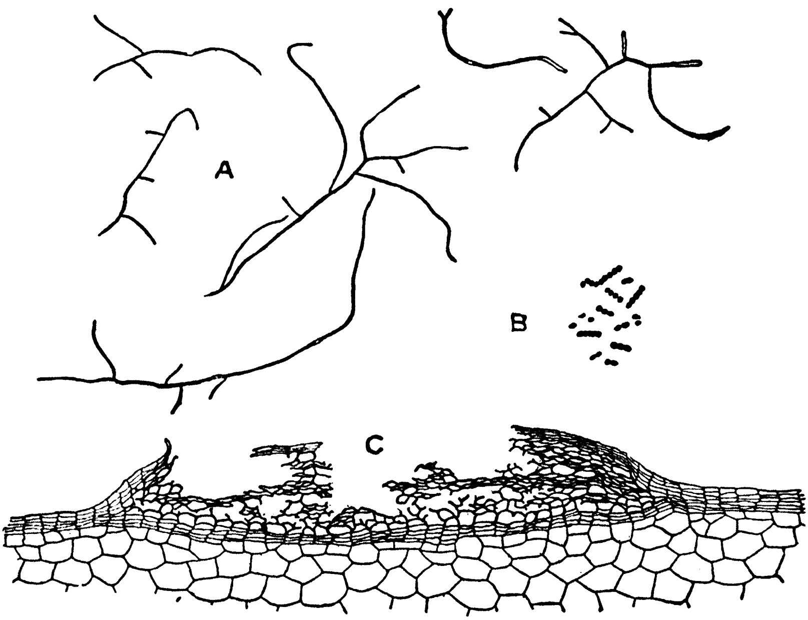

FIG. 103.—*A*, drawings of *Actinomyces scabies* showing branching and irregular segmentation; *B*, groups of gonidia; *C*, section of an old scab. (*After Lutman and Cunningham, Vt. Bul.* **184**.)

heap. Thaxter first showed that an infusion of horse dung was a good culture medium. Morse (1912) showed that the scab organism will survive passage through the digestive tract of domestic animals. In view of these facts it seems certain that the organism is spread by dumping scabby waste stock on the manure heap, and by feeding uncooked scabby potatoes to farm animals. As a result of analysis, a certain greenhouse soil was shown to contain 274,000 units, and a garden soil 466,000 units of *A. scabies* in every gram. Considering the wide distribution of the scab organism, it seems probable that the presence or absence of scab in severe form is dependent on the prevalence of environmental conditions of soil

moisture, temperature and soil reaction favorable or unfavorable to infection.

Predisposing Factors.—The natural soil reaction may be favorable or unfavorable to scab or this may be modified by the addition of fertilizers. Scabbing is slight in decidedly acid soils and the organism makes its best development in an alkaline or very slightly acid habitat. It has been shown by Gillespie (1918) that certain soils which have a pH exponent of about 5.2 generally produce a scab-free crop. In such cases the acidity is of sufficient intensity to exert an injurious action on the scab organism. The addition of *lime* has long been known to increase the amount of scab, as high as 40 per cent increase being noted. The effect is probably due in large part to the neutralization of the soil acidity. The use of *barnyard manure* favors scab, in two possible ways: (1) by the introduction of organisms in increased numbers; and (2) by augmenting the organic matter content and thus affording the most favorable conditions for the development of the scab organism. Even among the early writers, *wood* ashes are listed as aggravating scab, and their effect is supported by the work of later investigators. Potash salts, such as kainit, "almost invariably reduce or tend to control the ravages of potato scab," and sulphur, especially when applied to certain soils, is of value in reducing scab.

The amount of scab that develops is dependent on the soil temperature. According to Jones, McKinney and Fellows (1922) the optimum temperature is about 23°C. for the highest per cent of scabby tubers and 20.5°C. for the percentage of total tuber surface scabbed. The range of development of the disease is given as varying from 11 to 30.5°C., with but slight infections at either extreme. In certain field trials 25°C. was the most favorable temperature for scab development. These findings are in accord with the earlier report (Shapovalov, 1915) that the scab organism is favored by fairly high temperatures. It was found to make its best growth in pure cultures at temperatures ranging from 25 to 30°C. "Field observations seem in general to accord with the results obtained by experiments. They indicate that potato scab is comparatively more prevalent in regions having high summer temperatures than in those of lower temperature." The disease may be expected to vary in severity in a given region and to be least severe during the cool summers. The influence of soil temperature is undoubtedly an important factor in the reduction of scab in the Pacific Northwest, since the night temperatures are generally low even when the day temperatures are high.

The relation of soil moisture to the percentage of scab appearing in a crop has recently been investigated (Sanford, 1923). According to this report, "badly scabbed tubers were produced both in dry soil and in the medium (moist), but the potatoes in the moist soil were practically clean."

There can be no doubt that soil reaction, soil moisture and temperature are important factors in influencing the presence of and the degree of infection, but it has been pointed out that control has sometimes resulted, when there is no increase of acidity, with the use of either sulphur or green manure. It is suggested that the results may be due, in part, to the bactericidal action of sulphur and to the competitive action of stronger-growing, saprophytic Actinomycetes or other soil organisms.

Host Relations.—Corky scab is primarily a potato disease but it has been reported as affecting a number of other economic plants. It was recognized on beets as early as 1864, and Bolley (1893) showed that the scab of the beet was identical with that of the potato. Trials by Halsted indicated that beets, turnips and radishes were also susceptible, but no infections were obtained on the sweet potato, artichoke, salisfy and parsnip nor on a number of other less important plants. It is not exactly clear what part other hosts may play in perpetuating the scab organism, but it is certain that the pathogene may persist for years in the soil independent of any of the commonly recognized host plants.

Previous to the work of Lutman (1919) there had been numerous observations on the comparative resistance of varieties, but there seemed to be general agreement "on only a single point, namely, that some varieties of potatoes are more resistant than others." In the most extensive tests made, Stuart (1914) concluded that none of his 74 varieties "showed strongly marked scab-resisting qualities when grown on soil well infected with scab organisms" and also "that there seems to be little hope of securing scab-resistant varieties through selection." As a result of tests carried out through 4 years Lutman arrived at the following general conclusions: "Marked resistance to scab is found in the true russet types of tubers. The semirussets show some scab resistance while the white and thin-skinned varieties seem to be most susceptible." The per cent of clean tubers ranged all the way from 36 to 98 per cent in the most resistant varieties, and from 0 to 26 per cent in the most susceptible. Scab Proof, Burbank's Russet, Cambridge Russet and Dibble's Russet generally showed the greatest resistance, but there was much variation in the relative position of the more susceptible varieties in the different trials. These studies also led to the conclusion that "the thickness of the skin determines the resistance of the tubers to scab. Color seems to play no rôle in this resistance." It was also shown that "close-textured lenticels, partly buried under the skin surface and filled with small cells, are also associated with the russet type of potatoes," all varieties of which were shown to be at least moderately resistant. Various chemical treatments of the soil did not seem to modify the skin structure.

Prevention or Control.—There is no single measure which will control scab. The following practices have been shown to be of varying effi-

ciency, but only those should be selected which are suited to the particular environment or soil:

1. The use of clean or scab-free seed, preferably certified stock.

2. Crop rotation to avoid badly contaminated soils, using non-susceptible crops for at least 2 years.

3. Sanitary practices which retard or reduce soil contamination, such as: (*a*) exclusion of scabby stock from the compost heap; (*b*) the cooking of scabby tubers if to be fed to live stock; and (*c*) avoidance of fresh barnyard manure for fertilizer just previous to a potato crop.

4. Cultural practices which affect soil reaction or otherwise reduce infection: (*a*) avoidance of alkaline soils or practices which increase soil alkalinity such as application of lime or wood ashes; (*b*) the use of sulphate of ammonia instead of nitrate of soda if a complete fertilizer is needed on scab-contaminated soils; (*c*) the plowing under of a green cover crop such as rye before planting, especially where scab infestation is only moderate or slight; (*d*) the application of sulphur if the soil is heavily contaminated and rotation is impossible; and (*e*) delay in time of planting of early maturing varieties.

The following applications of sulphur have been recommended: 300 pounds per acre when the crop is only partially scabby; 500 pounds when crop has shown a high percentage of unsalable tubers, with a probable maximum of 400 pounds per acre for light, sandy soils. The application should be made with a lime distributor, just after the land is plowed, and should be harrowed in thoroughly at once. Under certain conditions *inoculated sulphur*,—that is, sulphur containing sulphofying organisms—has given better control than uninoculated sulphur (Martin, 1921), but in other tests there has been no appreciable benefit. Extensive tests of the value of sulphur have been made by Sherbakoff (1914), Lint (1914–1916), Martin (1920 and later) and Duff (1927).

5. Seed disinfection. The following treatments have been recommended: (*a*) corrosive sublimate either plain or acidified; (*b*) formaldehyde, either cold or hot; (*c*) copper sulphate, 3 pounds to 50 gallons, with immersion for 2 hours; (*d*) organic mercury preparations such as Semesan Bel or Bayer Dipdust; and (*e*) formaldehyde dust (Smuttox), on the basis of a single test only (Wiant, 1931).

Varying results have been obtained with these treatments, but the best success has followed the use of either corrosive sublimate or the hot formaldehyde, and both of these are about equally effective in the control of Rhizoctonia. The greater percentage of tests with the organic mercury compounds have not yielded satisfactory results. Because of their more frequent use with satisfactory results these two will be reported in more detail.

The hot-formaldehyde treatment has the advantage of a reduced time of steeping but the disadvantage of providing equipment for heating the bath. In this treatment the tubers are dipped for 2 to 3 minutes

in a bath of formaldehyde, 1 pint to 15 gallons of water at 118 to 122°F., after which they are removed, drained, and covered with wet sacks for 1 to 2 hours and then spread out to dry.

The following outline of the corrosive sublimate treatment, based on experience with Rhizoctonia, which is more difficult to control, may be presented: (*a*) Seed stock to be disinfected should be as free as possible from dirt, preferably *washed clean.* (*b*) Dip in water, drain and allow to stand in moist sacks for 12 to 24 hours. (*c*) Using barrels or wooden or concrete vats, dip the *whole tubers* in the solution of standard strength, in *open vats or crates* and not in burlap sacks: *For small lots of seed* (1 bushel or less) treat the first bushel 1½ hours, the second 1¾ hours, the third 2 hours, the fourth 2¼ hours and then discard the solution; *for larger lots of seed,* for each 4 bushels of clean stock treated by the standard formula, add ½ ounce of the chemical, corrosive sublimate, keeping the volume of the solution constant. Soak for 1½ to 2 hours. Discard the solution entirely after making six or eight treatments. (*d*) Treat preferably *before sprouting,* but at least 2 weeks before planting and dry the seed immediately after treatment. *Mercuric chloride is deadly poison,* so treated seed is unfit for food, should not be fed to live stock and special care should be given to the disposal of discarded solutions (see Rhizoctonia for use of acidified formula).

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IMPORTANT DISEASES DUE TO BACTERIA

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CHAPTER XIV

SLIME MOLDS AND PLANT DISEASES

MYXOMYCETES

Of the considerable array of known species of slime molds, none is now considered to be parasitic. The Plasmodiophoraceæ, including the forms responsible for the club root of crucifers and the powdery scab of potatoes, were formerly considered as belonging to the Myxomycetes, but present concepts of their relationship have put these forms with the Chytrids, as true fungi (see Chap. XVII). Brief consideration should, however, be given to the Myxomycetes, since certain species are frequently encountered in connection with growing crops and may exert injurious influences, although not in the manner of true parasites.

General Nature and Habitat of Slime Molds.—The slime molds, known first as the Myxogastres, and later as the Mycetozoa, are now generally called the Myxomycetes. They are a group of primitive organisms, standing on the border line between the plant and animal kingdoms, but scientists are now generally agreed that they are more plant-like than animal-like and related to the more primitive fungi. The slime molds are in the main saprophytic in their mode of life, living on decaying organic matter such as old logs, decaying stumps, fallen twigs or branches, leaf mold, compost heaps or other vegetable débris wherever there is sufficient moisture.

Vegetative Characters.—The plant body of the slime mold exhibits two distinct stages or phases: the *vegetative*, concerned primarily with nutritive activities, and the *reproductive stage*, which provides for the propagation and dissemination of the species. In its simplest condition the plant body consists of a simple, uninucleate, naked mass of protoplasm, amœboid in character, and therefore called a *myxamœba*. *Myxamœbæ* may increase in size by growth and become multinucleate, or several to many myxamœbæ may come together and form a larger, multinucleate mass of protoplasm, a *plasmodium*, the nuclei of which may divide and redivide and the whole structure increase in size. In many species the plasmodium is of microscopic size, while in others it may form large masses several inches across or spread over the substratum in branched sheets or net-like strands for a distance of even several feet. Young plasmodia may be nearly colorless in some cases, but the prevailing color of growing active plasmodia is yellow, while various shades of color may be exhibited. The plasmodium has about the consistency of the white of

an egg, and can creep from one moist substance to another during its active or vegetative stage.

Reproductive Stages.—A plasmodium may give rise to spores by the organization of simple or complex fruiting bodies, the *sporangia,* in which numerous uninucleate spores appear in dusty masses. The spores are small, 3 to 20μ in diameter, spherical or flattened by contact, smooth or rough, of various colors, but generally yellow or violet brown. The fruiting bodies show a great variety of forms from a flat, cake-like mass, an *ethalium,* to variously differentiated sporangia, spheroidal, elliptical or elongated, and sessile or stalked, with delicate thread-like elements of great variety of form and structure, the *capillitium,* mingled with or associated with the spores in the differentiated portion.

In the formation of the sporangia the external portion of the plasmodium is differentiated to form a structureless wall or *peridium* which encloses the spores and the *capillitium,* either in the form of an anastomosing network of cylindrical elements or as isolated cylindric elements with pointed ends, the *elaters.* Sometimes a part of the capillitial network may be grouped to form a denser structure, the *columella,* arising from the base of the sporangium or extending as a central strand. The capillitium functions in the dissemination of the spores by virtue of the strong hygroscopic properties of its threads.

Under suitable conditions of moisture the spores germinate when mature or soon after. The cell wall ruptures and the protoplasmic content escapes, either as a simple naked amœboid cell, essentially like an amœba, or the amœboid cell may be provided with a single, vibratile protoplasmic extension or cilium. These active cells or swarm spores may grow, and reproduce themselves by fission, or fuse in pairs, but finally they become the *myxamœbæ,* which grow into plasmodia or fuse to form plasmodia.

Two well-defined types of germination of the spores have been recognized: (1) the spore wall "ruptures by a deep, wedge-shaped split which widens, allowing the content of the spore to push out, as the skin of an overripe grape might split on pressure letting the pulp out"; (2) the spore wall is softened locally, probably by the action of an enzyme, allowing the swarm cell to creep out through a circular or jagged aperture (Gilbert, 1928). The number of swarm cells varies from one to four or, in Ceratiomyxa, to eight.

Under unfavorable conditions, such as extreme dryness, shortage of food or low temperature, the young amœbæ or swarm spores, at least in certain species, may pass into a resting condition, a *microcyst* or spore-like structure, to emerge later with the return of proper growing conditions. Under similar adverse conditions young plasmodia may behave in a similar way and form *macrocysts.* The resting stage of a mature plasmodium is called a *sclerotium.*

Classification.—Two subclasses of Myxomycetes are recognized:

Exosporeæ.—Spores superficial, developed on the outside of the fructification. This is represented by a single genus, *Cratiomyxa.*

Endosporeæ.—Spores developed within sporangia. This subclass includes numerous families and genera with about 500 recognized species.

Relation of Slime Molds to Crop Plants.—The slime molds live on decaying vegetable matter such as rotting leaves, wood, bark and vegetable débris in general and lead in the main a saprophytic life. In certain locations such as peat meadows, truck gardens, propagating frames or greenhouses, they may develop in sufficient profusion to cause some injury, mainly by interference with transpiration and photosynthetic processes. It would seem reasonable to expect such associations to lead to parasitism.

Only a few species have been recorded as causing injury to growing crops. These belong principally to the genera Physarum, Spumaria and Didymium, although others are occasionally mentioned. *Physarum gyrosum* has been reported as causing severe injury to asparagus (Flachs, 1927) while young cuttings of *Azalea indica* were killed and those of Camellia became chlorotic. *P. cinereum* has been very abundant in moorland meadows in Sweden (Wulff, 1906) where it occurred in small spots or strips sometimes 3 to 4 meters in length, covering the grass with a grayish coating of sporangia and later with the smutty liberated spores. *Spumaria alba* has been noted in abundance on clover, cucumber, strawberries and other garden plants (Flachs, 1927), and *Didymium annelus* on lettuce.

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CHAPTER XV

THE CONDITION OF A FUNGUS IN OR ON THE SUBSTRATUM

The Life Phases of Fungi.—Fungi like other plants show two phases in their life period. When a fungus begins growth on a given substratum its first activities are directed to the procuring (absorbing) of food and the building up of the fungous body, while the formation of specialized structures for the production and dissemination of new individuals follows as soon as the fungous body has obtained a certain size, vigor of development or age. The condition of a fungus in or on the substratum may be then either in one of the *vegetative stages* or the purely vegetative structures may have given rise to specialized *reproductive* stages or structures.

The object on or in which a fungus establishes itself may be designated as its *substratum*. This may be the humus of gardens or fields, the dead remains or products of animals or plants, an artificial medium, another living plant or animal or portions of such living organisms—in fact, any material which will furnish the necessary food.

Host and Parasite.—The plant pathologist is concerned primarily with fungi for which the substratum is a living plant or some organ or part of a living plant. The substratum may then be designated as the *host* (also *suscept*) and the fungus as the *parasite* which preys upon it. The fungus is simply carrying out its normal physiological processes—taking food from its host, growing and finally producing its reproductive structures. In this development it produces more or less serious disturbances in the life of its host, or disease in slight, pronounced or severe form is the result.

VEGETATIVE STAGES OR STRUCTURES

Hyphæ and Mycelia.—The typical fungous body consists of a delicate, branched, tubular or filamentous structure, the *mycelium*, microscopic in size or barely visible to the unaided eye. These mycelial threads or *hyphæ* may form an interlacing tangle, or a loose, woolly mass or they may be densely interwoven or even compacted into solid bodies. The hyphæ of a given fungus are either *septate* or *non-septate*, that is, divided by cross-walls or partitions at intervals or entirely devoid of cross-walls, and so distinctly tubular in character. Certain groups of fungi produce only non-septate hyphæ, while other groups exhibit only septate hyphæ of characteristic form and size. In working with fungi it is therefore of importance to determine first the septate or non-septate character of the

hyphæ. While the typical fungous body is of the nature described, certain fungi may consist of only a single globular cell no larger than individual cells of a hypha. These simple fungous bodies may represent

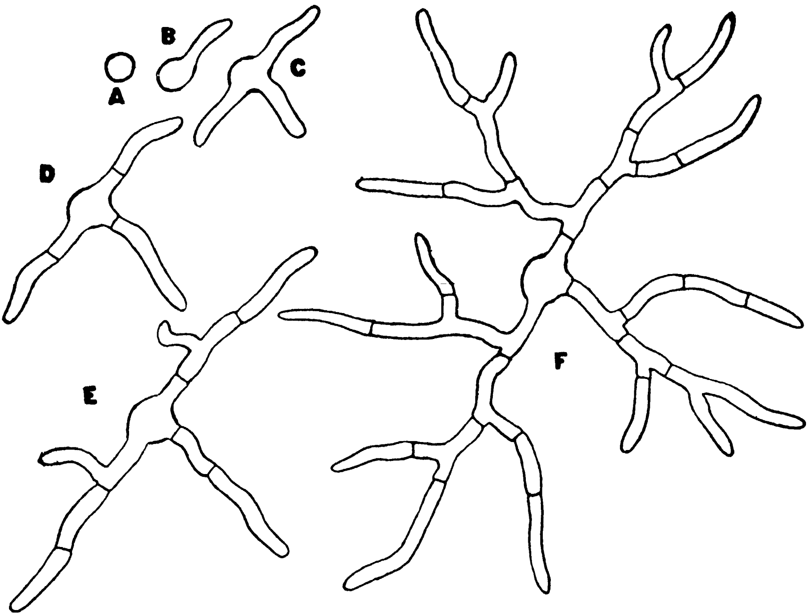

FIG. 104.—Outline diagram of a spore of Penicillium and stages in its germination to form hyphæ and a young septate mycelium. (*Adapted from Lafar.*)

primitive forms that have not yet organized a more complex plant body, or they may represent degenerates from more high specialized ancestors.

A parasitic mycelium may be either *external or internal*, that is, it may develop on the surface of its host, or it may grow within the tissues of its

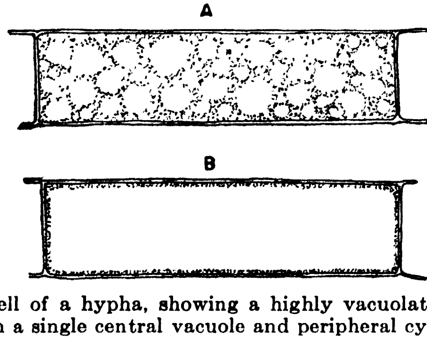

FIG. 105.—*A*, young cell of a hypha, showing a highly vacuolate cytoplasm; *B*, an old cell with a single central vacuole and peripheral cytoplasm.

host. External mycelia generally appear as delicate whitish, cobweb-like threads making an interlacing tangle or as sooty brown or black threads of similar character on the surface of leaves, stems or fruits. Such mycelia are very characteristic of the powdery mildews and the

sooty molds. Internal mycelia are confined to the intercellular spaces of the host tissue or penetrate into the interior of the host cells. These also may be clear or hyaline or smoky to dark brown in color. As they ramify through the host tissue, they are apparent to the unaided eye only when aggregated into dense masses. The presence of hyphæ within the invaded tissues of blighting leaves or rotting fruits can only be demonstrated by microscopic examinations.

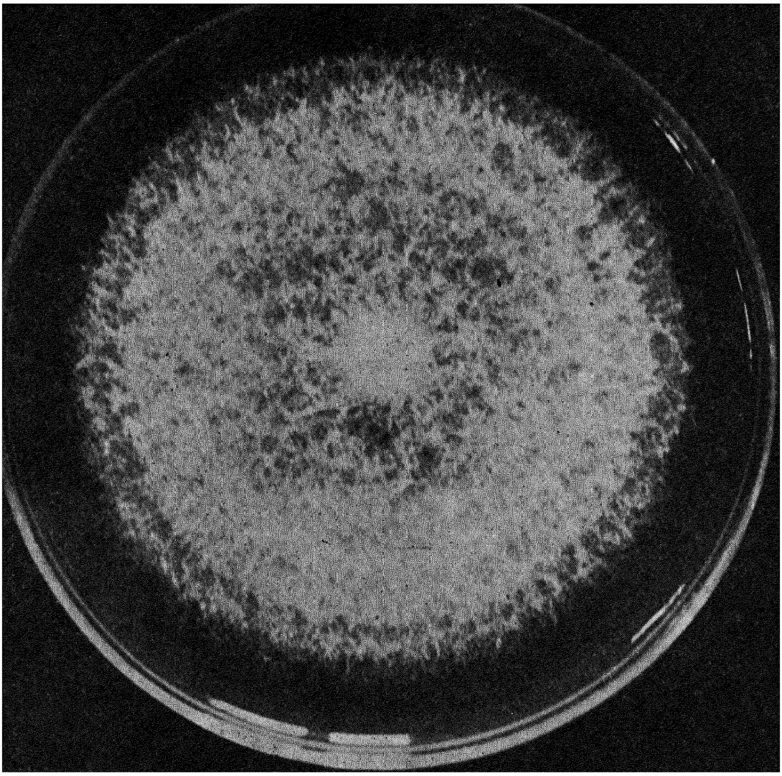

Fig. 106.—The mycelium of the silver-leaf fungus (*Stereum purpureum*) growing on agar.

When a mycelium begins to spread from a point or focus in which it is established, it shows a tendency to grow radially in all directions, and thus occupy an ever widening zone. This tendency is very frequently interfered with by the character of the substratum on which or in which the mycelium is developing. This peculiarity is manifested by both saprophytic and parasitic forms. Fungus "fairy rings" are the fruiting bodies developed on or near the periphery of an advancing mycelium, which is concealed within the soil. Many fungous leaf spots are distinctly circular, due to the manner of growth of the internal mycelium, while rotting areas on fruits show a circular surface outline for the same

reason. Fungous invasions of the bark of woody hosts generally develop slightly elongated or somewhat elliptical lesions or affected regions, due to the fact that the lengthwise advance of the mycelium is more rapid than the transverse growth.

Mycelial Plates or Fans.—In woody hosts and in some herbaceous hosts the mycelium becomes dense and compact and forms whitish pockets or radially or longitudinally elongated white plates or bands which are very evident when the invaded structures are cut open or broken. Such mycelial plates are especially characteristic of several

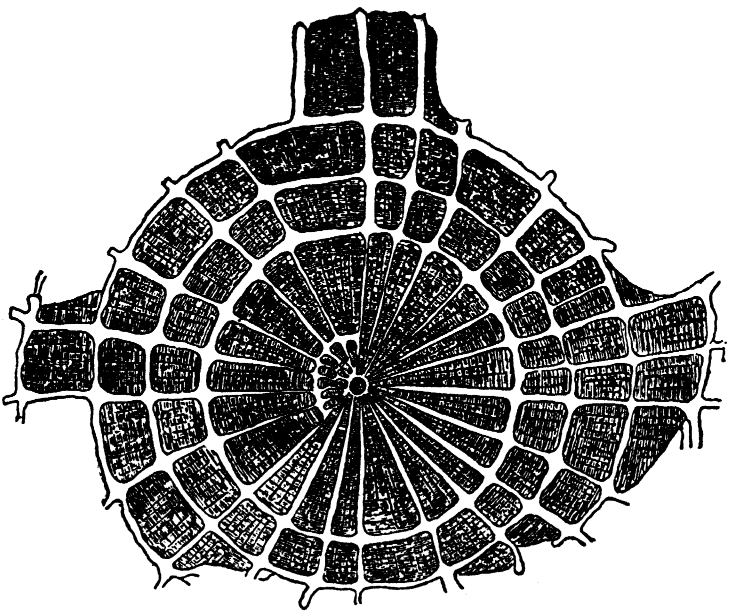

FIG. 107A.—Piece of timber infested with the mycelium of *P. sulphureus*. The white masses of fungus fill up the rings and rays produced by their "rotting" action. (*After Hartig.*)

rots of coniferous trees, such as the pines and spruces, where they occur within the disintegrating wood. The serious chestnut blight of the eastern United States is caused by a fungus which makes its greatest development in the inner layers of the bark or in the cambium, where very characteristic tawny sheets of mycelium spread out in fan-like forms.

Mycelial Strands or Rhizomorphs.—The hyphæ of a mycelium are sometimes aggregated in the form of cord-like or thread-like strands, which are in reality fungous cables. Such mycelial strands vary from slender threads to others that are equal in diameter to good-sized strings. The strands generally branch more or less and may frequently fuse to form a network. In some forms they are white and very conspicuous, while in other cases they are tawny or even dark brown.

The honey agaric which is responsible for the so-called mushroom root rot of numerous hosts, especially fruit and forest trees, is frequently called the shoestring fungus, because of its brown root-like strands which mingle with the roots of the host or run over their surface. Mycelium from these strands penetrates the bark and wood of roots or crown and causes the fatal disintegration or rotting of the tissues. On account of their root-like character these rhizomorphs frequently pass unobserved, but to one who becomes familiar with their appearance they offer a certain means of determining the presence of root rot in suspected cases.

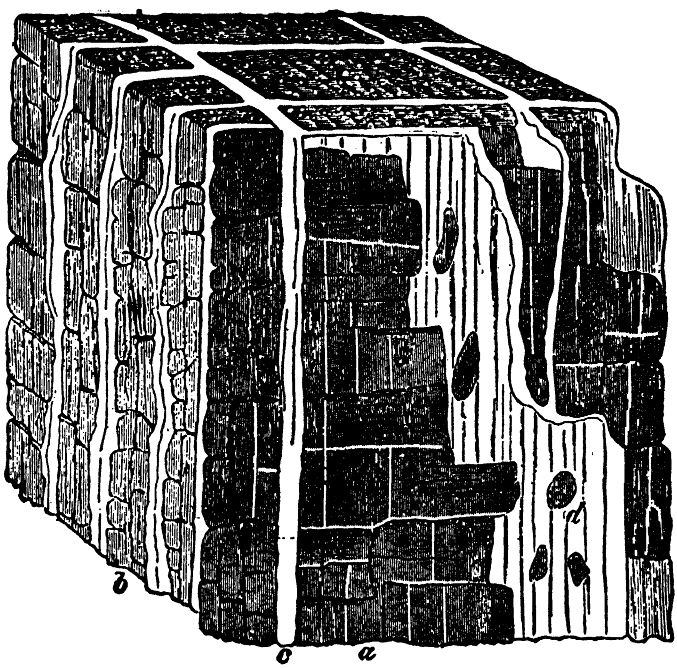

FIG. 107*B*.—Piece of timber completely destroyed by *P. sulphureus*, the mycelium of which fills up the crevices as a white felt. (*After Hartig.*)

While these fungous strands may store up some food, they are not primarily storage organs, but serve rather to bring the fungus to new hosts or to new parts of the same host and thus widen or enlarge the area occupied. In this way the fungus may spread from a single diseased tree to adjacent trees by a natural growth. It is undoubtedly true that in orchard practice fragments of rhizomorphs may be torn loose in cultivating and carried away to other parts of the field.

Sclerotia or Storage Organs.—Some fungi have developed the habit of producing dense compacted aggregates of hyphæ which become filled with food materials in the form of oil and other compounds. These structures vary in size from those that are hardly visible to the unaided eye to others that are as large as a cantaloupe or even larger, and are called

sclerotia, the term being derived from the Greek work meaning "hard." Sclerotia are generally more or less rounded, elongated, cylindrical, globular or ellipsoidal masses, but they are sometimes more or less flattened and irregular in form. In many cases they are dark colored, either on the surface or throughout the entire aggregate of cells. In

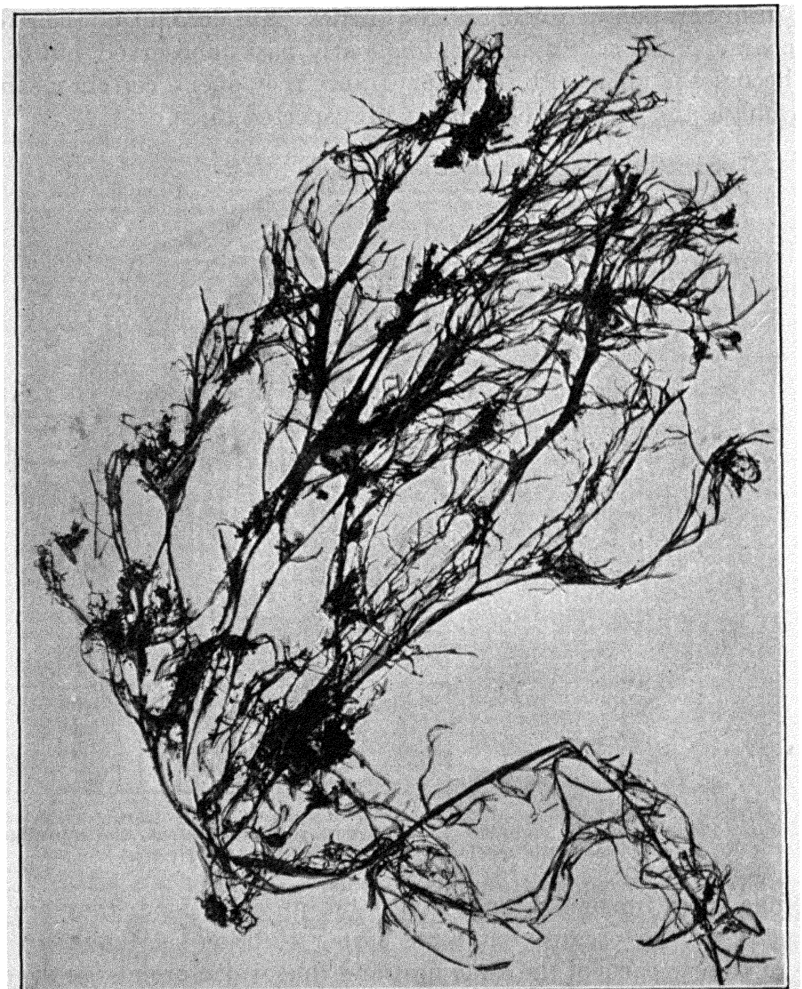

FIG. 108.—Rhizomorphs or fungous strands of the shoestring fungus (*Armillaria mellea*). (*After Freeman, Minnesota Plant Diseases.*)

parasitic fungi, they are formed either upon the surface of host parts, concealed between rotting leaves or within internal cavities. Sclerotial formation is not peculiar to parasitic fungi, but some saprophytes, like the carrion fungi and pore fungi, produce underground storage organs of considerable size. The so-called "Tuckahoe Indian Bread" of the southeastern states is a large sclerotium of the basidiomycete, *Poria cocos.*

A few examples of sclerotial formation by parasitic fungi which show interesting adaptations will be noted. In many regions potato tubers which show "dirt that will not wash off" are of very common occurrence. These brown or black, hard masses which are the size of a pin head or larger (maximum 1 inch) are the sclerotia of a Rhizoctonia, a fungus which is responsible for the Rhizoctonia disease of potatoes and many other hosts. Alfalfa, clover, beans and some other non-leguminous hosts when suffering from a disease known as wilt produce on the crown or roots, or within the pith cavity of the basal portions of the affected stems, rounded or elongated, seed-like bodies, black without but whitish within, which are the storage organs or sclerotia of the wilt fungi (*Sclerotinia spp.*).

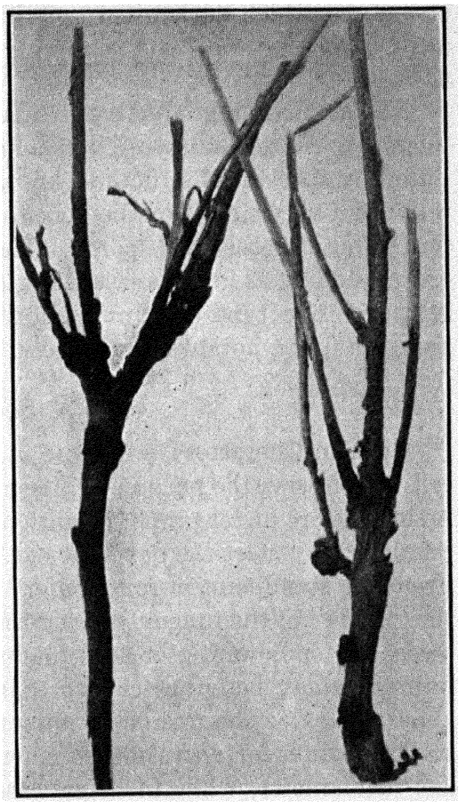

FIG. 109.—Sclerotia of the wilt fungus (*Sclerotinia trifoliorum*) on alfalfa plants.

The ergots of rye and other grasses are elongated, cylindrical, black or purple, horny sclerotia, which take the place of normal grains. Some fungi that are parasitic on insects show a very similar development. The fungus gains an entrance into the body of the insect, and in the caterpillar fungus fills up the entire larval body with a dense mass of fungous tissue which replaces the normal host structures. The larva is killed and the "fungus caterpillar" or cast takes its place.

Sclerotia are, in reality, storage organs filled with the special kinds of reserve food peculiar to the species of fungus by which they are formed. Although they are vegetative structures, they serve essentially the same purpose in the life of many fungi as spores, for they are able to withstand adverse conditions which would prove fatal to ordinary mycelia. In the sclerotial condition fungi are able to endure extreme desiccation, or long periods of high temperatures or the rigors of winter. Attached to host parts, such as seed or propagating stock, or mingled with the soil or seed, sclerotia are frequently very effective means of the dissemination of parasitic fungi.

There is great variation in the behavior of sclerotia. Those of Rhizoctonia reproduce the fungus by the production of new vegetative hyphæ when favorable conditions for growth are offered; the sclerotia of wilt and other Sclerotinias after passing through a winter period give rise to apothecial fruits, while the ergots of rye and other grasses develop new structures, which in their turn produce perithecia.

REPRODUCTIVE STAGES OR STRUCTURES

The common reproductive structures of fungi are very small bodies of microscopic size, the *spores*, which are either cut off from hyphæ or produced by specialized structures known as *spore fruits*. Some fungi have retained a very simple method of propagation which does not involve the formation of specialized spores. The production of new cells, by a process of budding as in the yeast plant, essentially similar to the parent cell, is a primitive type of reproduction, which is retained by some of our parasitic fungi, notably some smuts, at certain stages in their life cycle.

A. Spores

General Characters.—A spore is generally one to several specialized cells which serve the purpose of disseminating and reproducing the fungus. A single spore under proper conditions of temperature, moisture and host relations may start the development of a new fungous body, a *mycelium*. From the standpoint of function or the part which they play, spores are the "seeds" of the fungus, serving the same purpose for fungi as the true seeds do for flowering or seed plants. Spores vary greatly in size and form and may be either clear or dark, varying from slightly smoky to almost black. The micron or micromillimeter ($1/1000$ millimeter) is the standard unit for spore measurement, the smallest forms being 1μ or less in diameter, while in a few cases they may reach 1 millimeter or slightly more. In form they may be globular, ellipsoid, ovate, cylindric, filamentous, club shaped, star shaped, etc. (Fig. 110).

Sexual and Asexual Spores.—Spores may be formed as the result of a breeding act, that is, the union of two separate and distinct cells or elements (gametes) which represent male and female. Spores of this type may be classed as *sexual*, while those which are formed direct from hyphæ without the intervention of a breeding act are *asexual*, or without sex. It is undoubtedly true that asexual methods of spore formation were the most primitive, while sexuality was a later acquirement.

Kinds of Spores.—Considerable variation is shown in the structure and origin of spores and special names are used for the different kinds. *Chlamydospores* are formed by the direct transformation of certain cells of a hypha, without the production of specialized spore-bearing branches. Only scattered cells may be organized as spores (Mucor species) or the entire hypha may be used up in spore production (Smuts). *Swarm spores*

or *zoöspores* are naked protoplasmic masses provided with delicate vibratile filaments or thread-like processes, the *cilia*, by means of which they are able to swim about. It is this power of locomotion which has suggested the name zoöspore, or animal spore. The cilia, or organs of locomotion, are variously arranged, different species or groups of fungi showing one, two or a tuft of polar cilia, or they may be numerous and distributed over the entire surface of the spore. Certain swarm spores show a very characteristic kidney shape, with two cilia originating at the suture. All other spores are provided with a definite cell wall or membrane, and in many forms it is variously modified or thickened.

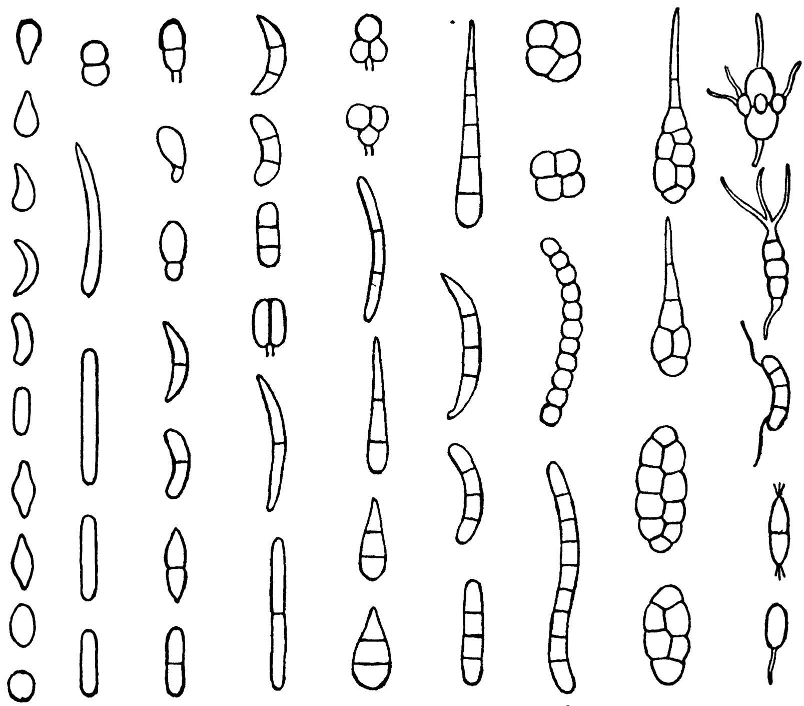

FIG. 110.—Diagrams of various forms of spores.

Conidia is a general term applied to spores which are pinched off or cut off from the ends of special spore-bearing hyphæ, known as *conidiophores* or conidia bearers. Such spores may accumulate in clusters or in chains, and when in chains the terminal spore of the series may be the oldest in one case, while the basal spore is the oldest in others. There are many different kinds of conidia. *Ascospores* are produced within a sac-like or club-shaped structure, the *ascus*. In a young ascus there are two nuclei which soon fuse to form a single nucleus. In a typical ascus this nucleus soon divides into two, each dividing again to produce four, while the number is again increased by division, the final number being eight. Each one of these nuclei organizes a spore by surrounding itself

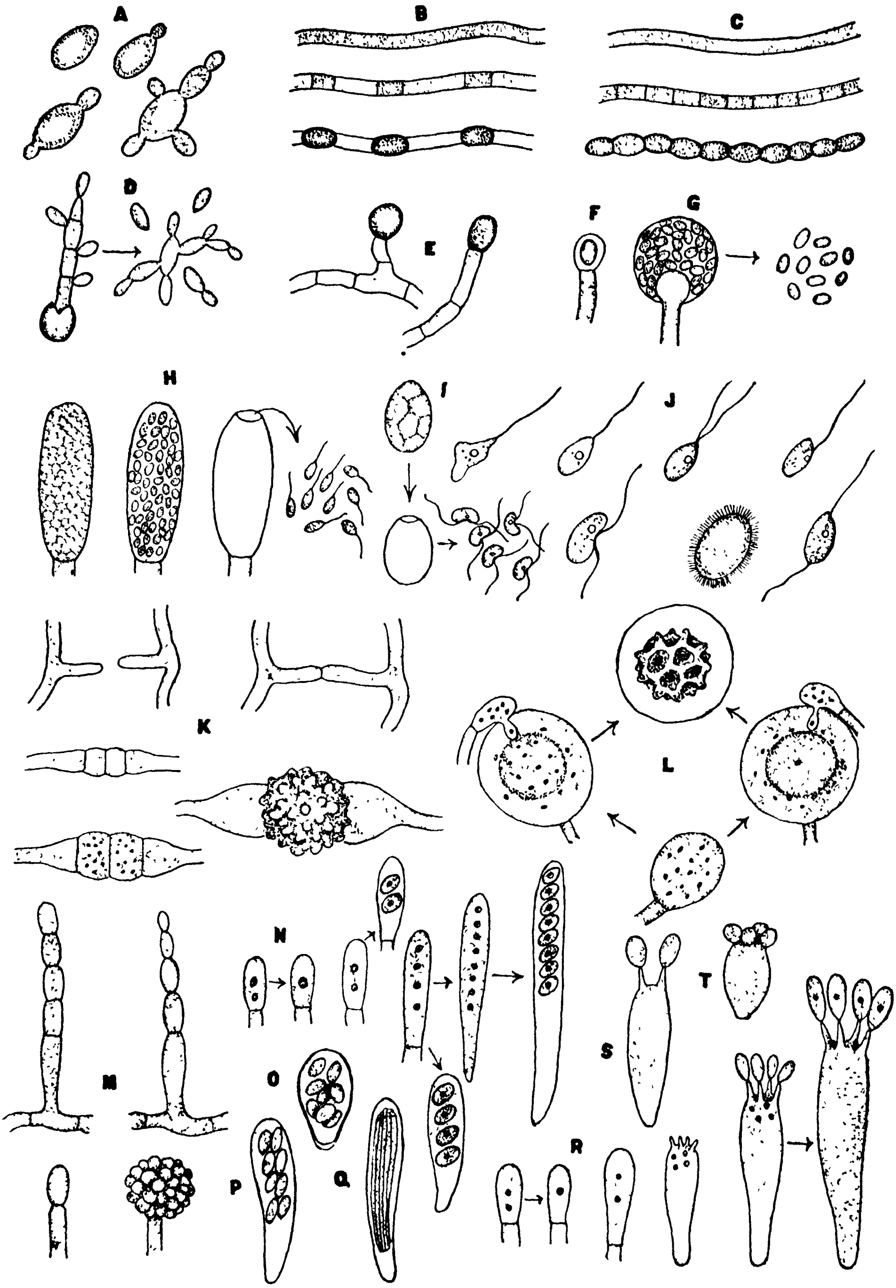

FIG. 111.—Semidiagrammatic drawings showing types of spores and their manner of formation. *A*, yeast cells, showing primitive method of propagation by budding; *B*, formation of chlamydospores; *C*, chlamydospores formed in a continuous chain; *D*, yeast-like method of production of secondary spores characteristic of some higher fungi, *e.g.*, smuts; *E*, chlamydospores produced at the ends of hyphæ; *F*, a sporangium bearing a single non-motile spore; *G*, a globular sporangium (Mucor type) with numerous non-motile spores; *H*, stages in the formation of uniciliate swarm spores from a sporangium;

and some of the adjacent ascus contents with a protecting cell wall. The result is a definite number of ascospores enclosed within the parent cell. There may be a deviation from the typical number of spores in some cases, one, two, four or sixteen being the common exceptions. Where there is an odd number of spores in an ascus it is due to the degeneration of one or more of the nuclei. The production of ascospores is characteristic of one great group of fungi, the ascomycetes or sac fungi. In certain of the higher fungi, like toadstools, shelf fungi, puff balls and related forms, spores are produced on structures very similar to asci in form, but called *basidia.* The spores, the *basidiospores,* instead of being produced within the structure, are developed on the tips of either two or four (rarely more) slender terminal projections (sterigmata). The number of spores is always definite and limited, a single spore being produced on each sterigma. In certain forms the basidia differ from the typical form by being divided lengthwise into four cells, each of which bears a spore, or they may be filamentous and divided transversely into four cells, each of which forms a spore. This later condition prevails in the true rust fungi, the basidium being designated as the *promycelium* and the spores as *sporidia.*

A spore formed by the union of two equal and similar gametes is called a *zygospore.* This is well illustrated in certain species of black molds. In bringing about this union or *conjugation,* adjacent hyphæ produce lateral branches, which develop in pairs, separate off a terminal multinucleate gamete from each, which fuse and form a thick-walled spore. In this case the two gametes which unite are equal in size and similar in behavior, so that male and female cannot be recognized. These gametes are, therefore, designated as positive and negative. Two positive gametes or two negative gametes would not have affinity for each other and could not unite to form a spore.

When the gametes which unite are of unequal size, the resulting spore is called an *oöspore* and the union is designated as *fertilization.* The large gamete, the female cell, is passive, while the small gamete, the male cell, is active. This type of spore formation is well illustrated in the white rust of Crucifers (see description under this disease). In the formation of both zygospores and oöspores nuclei of positive and negative gametes, or male and female gametes, fuse as the final act in conjugation or fertilization. Both zygospores and oöspores are thick-walled resting spores which may carry the fungus over an unfavorable period.

The special types of spores peculiar to the true rust fungi will be reserved for consideration in the treatment of that group.

I, a zoösporangium from which biciliate swarm spores have been formed; *J*, types of swarm spores; *K*, stages in the development of a zygospore from the union of equal and similar cells (gametes); *L*, two types of oöspore formation characteristic of white rusts and downy mildews; *M*, four types of origin of conidia; *N*, stages in the development of asci and ascospores; *O*, *P*, *Q*, three asci showing different arrangement of the ascospores; *R*, stages in the development of a basidium and basidiospores; *S*, *T*, two other types of basidia.

B. Spore Fruits

General Characters.—In the simpler forms of spore production, the spores are cut off from the ends of specialized, free aerial branches or conidiophores, or organized within the interior of specialized cells called *sporangia,* these spore-bearing branches being produced direct from the mycelium. With further development there is the organization of definite complex aggregates of spore-bearing hyphæ, frequently surrounded by more or less supporting and protecting tissue. It is these complex aggregates which can with real propriety be designated as *spore fruits,* since they are highly specialized structures adapted to spore production, protection and dissemination. Aerial conidiophores and sporangia represent a more primitive condition before the organization of the more complex spore fruits.

Kinds of Spore Fruits.—*Aerial conidiophores* are special branches of the mycelium set apart for spore production. They may be simple or variously branched, single or in groups or tufts and extend upward from the substratum so as to facilitate the separation and setting free of the spores. This type of spore production is very characteristic of the summer or conidial stage of powdery mildews, certain downy mildews and many forms of imperfect fungi.

The aerial conidiophores may arise singly from vegetative hyphæ either on or within the substratum. In the case of leaf parasites it is not uncommon for one or more to emerge to the exterior through a stomatal opening, thus forming conidial tufts or fascicles. Each spore-bearing branch may give rise to a single spore only or an indefinite number may be produced, which become grouped in chains or clumps or are detached as soon as mature. When chains of spores are formed, either the proximal one or the distal one may be the youngest. A conidiospore may be cut off from the end of the conidiophore by a cross-septum, or it may develop as a bud-like outgrowth from the conidiophore or from another spore.

In some of the imperfect fungi dense fascicles of erect conidiophores may be grouped together, thus producing a fruiting body known as a *coremium,* composed of a sterile stalk of parallel hyphæ and a terminal head of fertile or spore-bearing branches. This is the common or normal type of fruiting body for some fungi, while in others it appears to be induced by environmental conditions.

Sporangia are generally borne on aerial hyphæ which bear the same relation to the substratum as conidiophores. The special cell which is destined to form spores is separated off from the free end of the hypha by a cross-wall and organizes within its interior an indefinite number of spores, which are later set free by the breaking of the sporangial wall or through a special opening. In strictly aquatic fungi the sporangia form

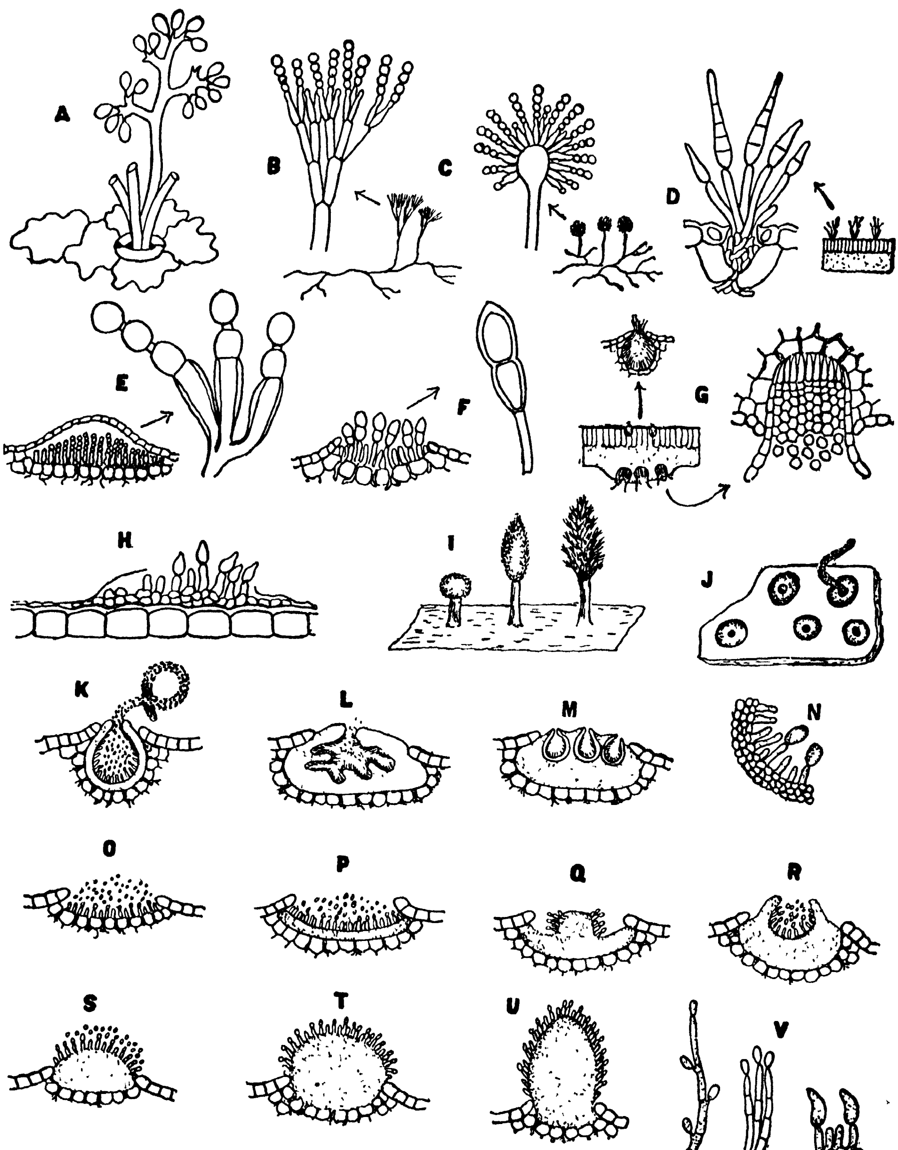

FIG. 112.—Semidiagrammatic drawings of types of conidiophores and spore fruits. *A*, a downy mildew, Peronospora; *B*, blue mold or Penicillium; *C*, Aspergillus; *D*, Cercospora; *E*, section of a sorus of white rust (Albugo) with enlarged conidiophores and conidia; *F*, section of a telium or teleutosorus of a true rust with a single enlarged teliospore; *G*, section of cluster cups or æcia and pycnia (above) with each spore fruit enlarged; *H*, conidiophores and conidia of the apple-scab fungus; *I*, three types of coremia; *J*, a habit sketch of pycnidia; *K*, section of a pycnidium with a spore tendril protruding from the ostiole; *L*, section of a stromatic pycnidium; *M*, section of a stroma with several immersed pycnidia; *N*, detail of small portion of the wall of a pycnidium showing conidiospores and conidia or pycnospores; *O–R*, types of acervuli; *S–U*, types of sporodochia; *V*, three different types of conidiophores from spore fruits.

swimming or *swarm spores* adapted for life in the water, while terrestrial species may form only non-motile spores adapted to air or wind dissemination. Some terrestrial species very closely related to aquatic ancestors have retained the swarm-spore habit—for example, the white rusts and some of the downy mildews.

In certain fungi the spores are grouped in small or large masses or clusters to which the name *sori*, which really means "little heaps," is applied. The whitish spore dots or pustules of the white rusts, and spore dots or pustules of the red or black rust stages of the true rusts, are designated as sori, while the term is also applied to the much larger, black, powdery spore masses of the smut fungi. A single sorus is a group of either spores alone or spore-bearing hyphæ or stalk cells with spores which may sometimes be mingled with sterile filaments. Sori may be naked or covered, that is, they may remain covered with some of the host tissue, or the spore masses may be exposed to the surface so as to appear granular or powdery. The red powder which is abundant on the leaves and stems of badly rusted wheat or oats or other cereals originates from numerous sori which break out through the host tissue. The smut dust which comes from a threshing machine working on heavily smutted grain is due to the breaking up of the smut masses or sori, the dust particles being either single spores or groups of spores. Many of the true rust fungi also produce small, flask-shaped fruiting bodies, the *pycnia* or *spermogonia* embedded in the host tissue, generally leaves. These appear on the surface as minute specks or pimples barely visible to the unaided eye. The inner wall is lined with numerous hyphal branches which separate off minute, bacteria-like bodies known as *pycniospores* or *spermatia.*

Certain species of the true rusts form another type of fruiting body which appears soon after the *pycnia* and generally in connection with them. In the most typical form of these *æcia* or *æcidia*, there is a mass of closely packed, yellow- or orange-colored spores borne within a cup-like structure composed of specialized but sterile fungous cells. The surrounding membrane may be toothed, lacerate or cut into long lobes, irregular or entirely absent. The æcia are generally produced in groups or clusters, which with the cup-like form has suggested the name "cluster cup," which is frequently applied to this spore stage. The *æciospores*, when mature, readily rattle out of the little cups and are borne away by air or wind.

The *pycnidium* (pycnid for short) is a type of fungous fruit that is peculiar to certain imperfect fungi or may occur in the life cycle of some sac fungi. In typical form it is a more or less globular structure, embedded in the substratum and opening out to the surface by a pore known as the *ostiole.* The wall of the pycnid generally consists of one to several layers of fungous cells with simple or branched conidiophores lining its inner surface. Spores are produced in large numbers from these

conidiophores and either accumulate within the pycnid or are forced out through the ostiole. These *pycnospores* frequently accumulate in sticky masses over the pycnid or are forced out in long coils or tendrils. These tendrils or "*spore horns*" vary from those of microscopic size to others an inch or more in length. These spore threads are frequently hard and horny when dry, but when soaked by rains become soft and permit the separation and washing away of the spores by solution of the mucilaginous matrix in which they are embedded. Pycnidia are generally minute structures, barely visible to the naked eye as pale, smoky, black or even colored bodies immersed in the substratum or resting on its surface. They may be embedded in an aggregated mass of fungous tissue known as a *stroma.*

The *acervulus* (plural acervuli) is a type of spore fruit that is peculiar to the Melanconiales, a group of imperfect fungi, and appears in the life cycle of some sac fungi. In typical form it consists of a saucer-shaped, depressed structure which bears conidiophores over its exposed surface and sets the spores free by rupture of the superficial cell layers of the host. Acervuli are comparable to pycnids in size and in the manner of spore production and liberation. The basal matrix which bears the conidiophores, and is comparable to the pycnidial wall, may be well developed and very evident, or it may be almost lacking.

Certain other conidial fruits, the *sporodochia* (singular, sporodochium) are very similar to the acervuli, except that there is a development of a pronounced stroma-like cushion of fungous tissue, which breaks through the host tissue and bears conidiophores over most of its exposed surface. Sporodochia may also be seated in a mass or tangle of mycelium, which has become superficial. The spores may accumulate in sticky masses or they may be dry and powdery.

In some of the more primitive sac fungi, the asci or spore sacs are arranged side by side in an extensive layer over the surface of host parts without being collected in definite fruiting bodies (leaf-curl fungi). A closed ascus or ascigerous fruit, the *cleistothecium* (also called a perithecium) is characteristic of the powdery mildews. It is developed immediately following the union of male and female gametes, the product of the union growing at once into a new structure rather than organizing a definite resting spore. The ascus fruits are borne in the superficial mycelium and appear as minute, globular or slightly depressed black bodies easily visible to the unaided eye. A single fruit consists of a surrounding envelope of brown, sterile fungous cells which completely encloses one or more asci. Sterile threads or hyphæ known as *appendages* extend out from the cleistothecium wall and exhibit characteristic forms in different generic types. The ascospores are not discharged until the warm spring rains, when the wall of the spore fruit ruptures and exposes the asci which burst and forcibly eject the ascospores.

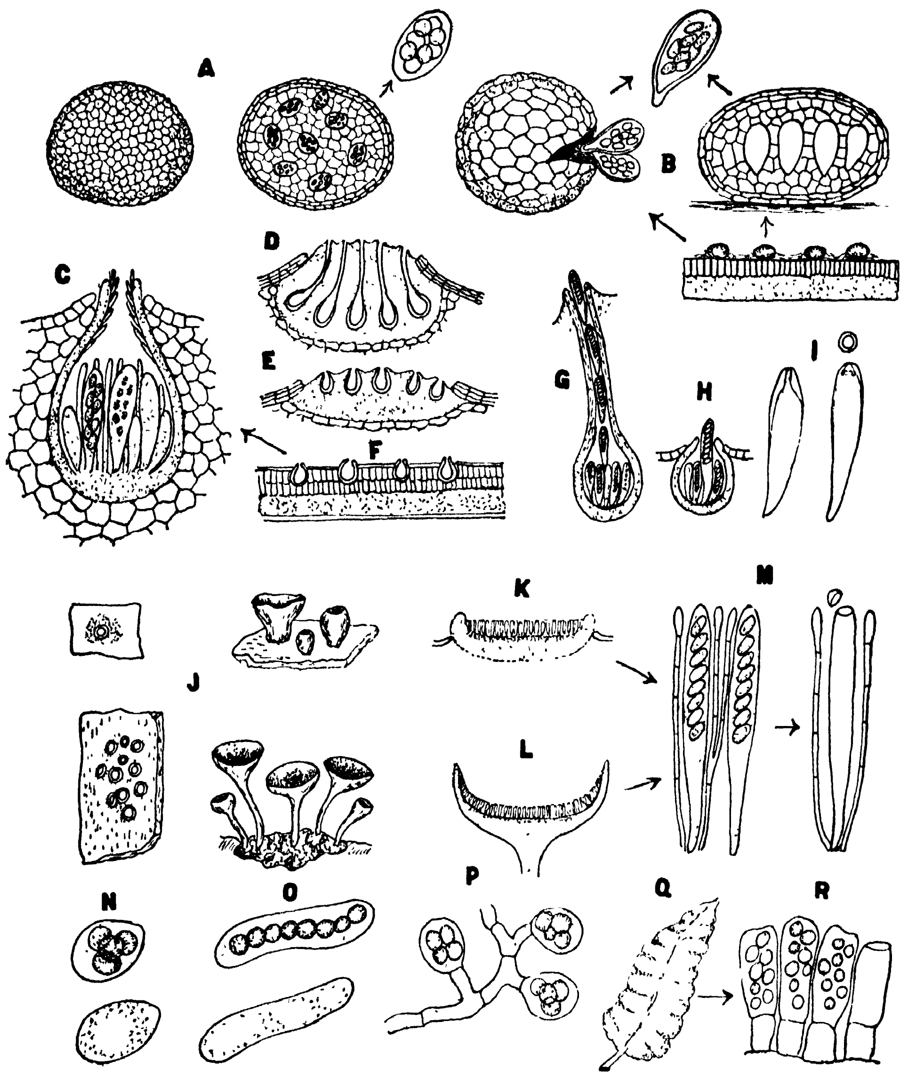

FIG. 113.—Semidiagrammatic drawings of ascigerous fruits and simple ascus-forming fungi. *A*, surface and sectional view of a closed ascocarp of Aspergillus, with a single enlarged ascus; *B*, habit sketch, surface view with escaping asci, enlarged ascus and section of a perithecium of a powdery mildew; *C*, vertical section of a typical ostiolate perithecium; *D* and *E*, sections of stromata with immersed perithecia; *F*, habit sketch of typical perithecia; *G*, section of a perithecium with long neck showing how the asci are released and forced up to the ostiole for the discharge of spores; *H*, section of a perithecium showing the manner of elongation of asci through the ostiole for the expulsion of the ascospores; *I*, two types of asci, one with terminal sphincter, the other terminal canal, structures used in spore discharge; *J*, four types of apothecia or fruits of cup fungi; *K*, section of a sessile apothecium; *L*, section of a stalked or stipitate apothecium; *M*, asci and paraphyses, or sterile filaments from a typical apothecium, showing one empty ascus with the lid separated; *N*, *O*, vegetative yeast cells and sporulating cells or simple asci; *P*, asci of a simple filamentous fungus, Endomyces; *Q*, leaf of peach affected by leaf curl, or *Taphrina deformans; R*, asci from the surface of the same leaf.

The typical *perithecium* (plural perithecia) resembles a pycnidium in the form, size and character of its wall and exhibits a similar opening or ostiole. It is then a more or less flask-shaped body of fungous tissue enclosing a group of elongated or club-shaped asci which arise from a basal aggregate of cells. Mingled with the asci there may be sterile slender filaments, the *paraphyses*, but these structures are not always present. Perithecia may be borne single or in closely associated groups, either immersed in host tissue or superficial, or they may be aggregated in compact masses of fungous tissue, or *stromata*. Some forms show no definite perithecial walls but appear as cavities in an undifferentiated mass of parenchyma-like tissue. Ascospores are sometimes set free from the perithecium in sticky or gelatinous masses, but in most of the parasitic species they are forcibly expelled. In a typical case an ascus elongates and pushes its tip through the ostiole. A strong hydrostatic pressure is developed within the ascus, and this finally ruptures the ascus wall or forces an opening. With the sudden release of pressure the charge of ascospores, eight in number, is shot out into the outside air. The old ascus wall then collapses and another ascus is pushed up to the ostiole and the process of spore discharge is repeated. This expulsion then continues until all of the asci have liberated their spores.

The *apothecium* is a third type of ascus fruit and in its typical form consists of a disk-like, saucer-shaped or cup-like body, seated on or in the substratum or raised on a long or short stalk or stipe. The exposed flat or concave surface consists of an extended layer of cylindrical asci, closely packed side by side (the hymenium) and generally mingled with more slender sterile hyphæ, the paraphyses. The wall of the disk or cup is made up of supporting fungous tissue, filamentous in character or of closely packed cells (pseudoparenchyma). Apothecia vary from minute structures barely visible to the unaided eye to others several inches in length or diameter, and are usually more or less fleshy in character, which is in contrast to the firmer texture of perithecia. Some of the saprophytic fungi produce greatly modified apothecia, as illustrated by morels, saddle fungi and their allies. In these forms the cup has been practically turned inside out, and in the morels shows a greatly convoluted hymenial surface, giving much larger space for the display of asci, while in the saddle fungi the enlarged cup is folded so as to suggest the common name. In the truffles the cup remains closed and develops underground, never coming to the surface to discharge its spores. In the great majority of apothecia the ascospores are set free by explosion of the asci. In these, however, there is a simultaneous discharge of numerous asci, with a resultant cloud of spores generally visible to the naked eye. This phenomenon is spoken of as the "puffing of spores," and serves to separate the spores from the fruiting body and to facilitate effective air or wind dissemination.

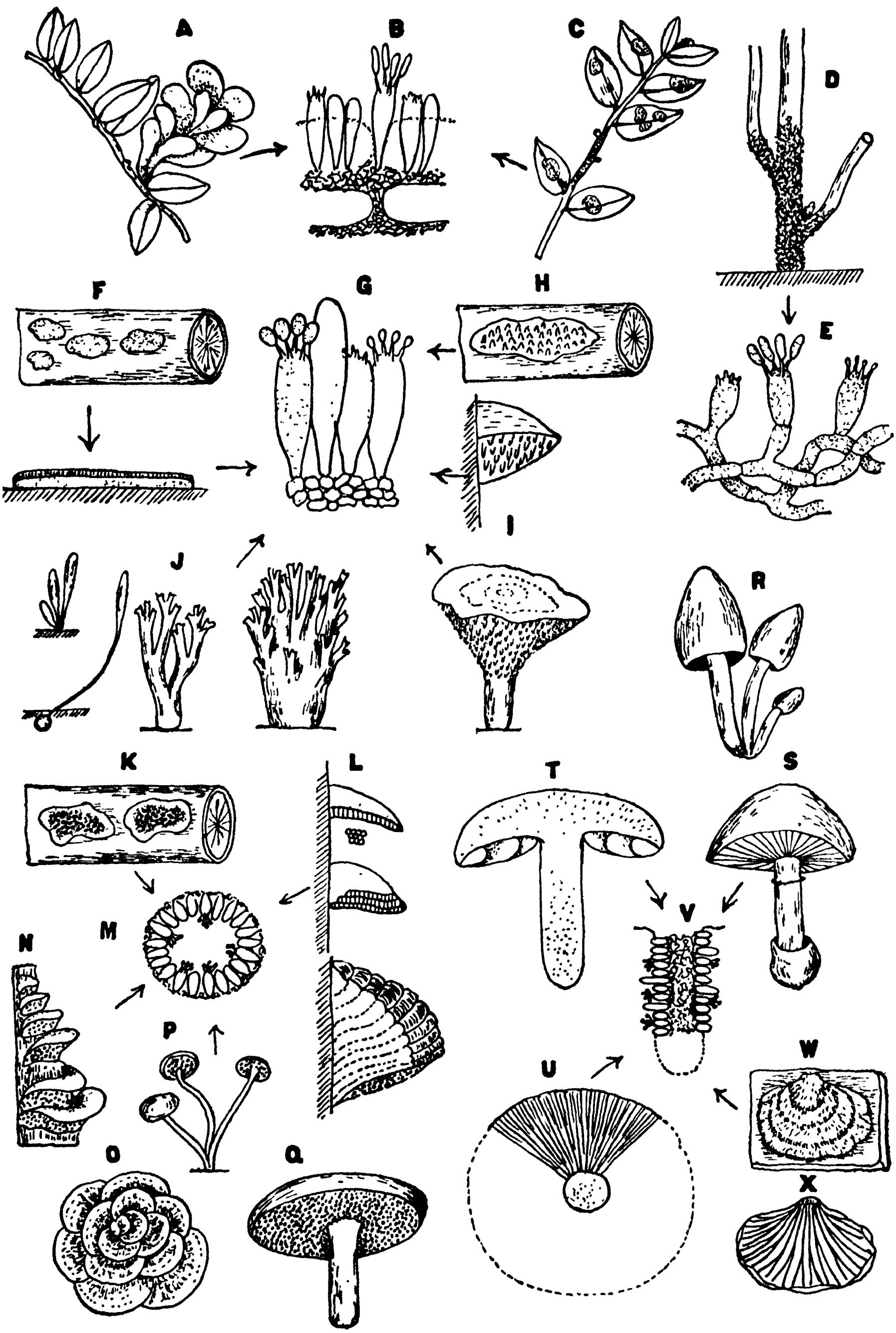

FIG. 114.—Semidiagrammatic drawings of basidium fruits. *A*, cranberry affected with rose bloom (*Exobasidium oxycocci*), showing enlarged flower-like lateral shoot; *B*, basidia from the surface of one of the hypertrophied leaves shown in *A*; *C*, red leaf spot of the cranberry, with leaf and stem lesions which produce basidia similar to those shown in Fig. *B*; *D*, base of stem showing a simple basidial felt (Hypochnus or Corticium type);

In some of the more primitive basidium fungi there is no organization of a definite fruiting body, the basidia being produced from the general mycelium which develops on or within the substratum. In certain parasitic gall-producing forms (Exobasidiales) the basidia originate from an internal mycelium and are produced over the surface of the enlarged and modified structures, the relation being very similar to that of the asci to the mycelium in the leaf-curl fungi. The production of spores gives a whitish, powdery appearance to the basidial surface. In some of the mold-like basidium fungi, the basidia are borne in irregular clusters or groups over the surface of a superficial mold-like growth. This is the condition which prevails in *Corticium vagum,* the cause of the Rhizoctonia disease of potatoes and other hosts when the basidium stage appears as a whitish, powdery, mold-like covering on the surface of the stem just above ground level.

In the great majority of basidium fungi, the basidia are grouped and supported by rather complex structures which may be designated as *compound sporophores.* These sporophores are of varying texture and may be fleshy, leathery, horny or woody. Some are small but they are generally large in comparison with other types of fungous fruits, the greatest size being reached in some of the giant puff balls and monstrous bracket or shelf fungi. The prime purpose of the sporophore is to afford a surface for the support and display, and in some cases for the protection, of the basidial layers, and the surface is increased by various structural modifications. The simplest sporophores are simply prostrate layers of fungous tissue with a smooth, plain basidial surface. Other forms may be simple clubs or compound clubs and more or less coralloid, while others are of the toadstool form or still others bracket-like or shelf-like in character. Puff balls represent a still different modification of the basidium fruit, with basidia developed entirely within the closed fruit. The "smoking" of puff balls when squeezed is due to the liberation of innumerable numbers of basidiospores. In the different forms of sporophores the basidial surface may be increased by wrinkles, ridges, tooth-like projections, thin plates or lamellæ packed closely together, a honeycomb-like structure or minute parallel tubes which appear as pores on the under surface of the sporophore.

E, hyphæ and basidia from *D*; *F*, habit sketch and section of a plain resupinate sporophore (Stereum type); *G*, typical basidia with one sterile cell or cystidium; *H*, *I*, resupinate, shelving and stalked sporophores with toothed basidial surfaces (Hydnum type); *J*, sporophores of fairy clubs and coral fungi (Clavaria type); *K*, habit sketch of a resupinate. poroid sporophore; *L*, sections of annual and perennial bracket sporophores (Polyporus and Fomes types); *M*, section through a single pore showing arrangement of the basidia; *N*, *O*, imbricated bracket fruits of the pore type; *P*, *Q*, stipitate or stalked sporophores, the basidia in pores; *R*, *S*, sporophores of gill fungi or toadstool forms (Agaricus type); *T*, section of a toadstool form through the middle of the cap or pileus and the stipe or stalk showing the varying lengths of the basidium-bearing plates or lamellæ; *U*, arrangement of the gills as viewed from the under surface of the pileus; *V*, section of a portion of a gill showing arrangement of the basidia; *W*, upper surface of the sporophore of a common gill fungus (*Schizophyllum alneum*); *X*, under surface of the same sporophore showing the arrangement of the gills or lamellæ.

But very few of the club or coral fungi or the puff balls furnish parasitic species, but there are numerous parasitic species which illustrate the other types of sporophores. The basidium fungi are the principal wood-destroying forms and may produce either root or trunk rots.

Reference

See textbooks listed at the end of Chap. I.

CHAPTER XVI

DISEASES DUE TO DOWNY MILDEWS AND ALLIES

OÖMYCETES

The parasitic fungi which form a non-septate mycelium and reproduce by the development of oöspores, unless sexual spores are omitted, and by either swarm spores or conidia (the Oömycetes) may be placed in the three following orders:

1. **The chytrids** (Chytridiales) with a plant body consisting mostly of a single unbranched or only slightly branched cell. While this order includes many species which are parasitic upon algæ, it also furnishes a number of very important pathogenes of our crop plants (see special treatment of Diseases Due to Chytrids, Chap. XVII).

2. **The water molds** (Saprolegniales) with well-developed mycelium, principally saprophytic in habit. Many of the species may be found in the fresh waters of streams or lakes living upon dead insects, fish or plant remains, while others are common in the soil. A few forms are parasitic on fresh-water algæ, fish or other aquatic animals.

Asexual reproduction is by biciliate swarm spores produced in specialized sporangia which are mostly persistent. Sexual reproduction is by antheridia and oögonia containing one to several naked oöspheres or egg cells. The order comprises two families:

The Leptomitaceæ including six genera, all species saprophytic. One species, *Leptomitus lacteus*, is noteworthy because of its development in drain pipes and sewer water.

The Saprolegniaceæ including fourteen genera, of which only one, **Aphanomyces,** is of importance as furnishing destructive plant parasites. **Plectospira** is of minor importance.

3. **The pythiaceous fungi, white rusts and downy mildews** (*Peronosporales*), which are principally parasitic in habit, and for the most part obligate parasities. The chief distinguishing features of this order are: (*a*) a well-developed non-septate mycelium which is intercellular, with the exception of forms of Pythiaceæ, which are also intracellular; (*b*) asexual reproduction by *conidia* (*zoösporangia*), which in the more primitive forms produce *swarm spores* and hence are sporangia, while in the highest types an infection hypha is the first product of germination; and (*c*) sexual reproduction, when this is not suppressed, by the union of unequal and dissimilar gametes to form *oöspores.* A large female cell or *oögonium* produces either a uninucleate or multinucleate gamete, while the

male cell or *antheridium* is smaller and gives rise to several male gametes or sperm nuclei. The three following families are recognized: *Pythiaceæ, Albuginaceæ* and *Peronosporaceæ.*

PYTHIACEÆ

This family represents transitional forms between the water molds (Saprolegniales) and the downy mildews (Peronosporaceæ). They have specialized in the development of zoösporangia. In the more primitive forms definitely differentiated conidiophores are lacking, the swarm sporangia being formed from ordinary hyphæ, to which they remain attached. Aerial conidiophores, when formed, may be delicate or robust, and either simple or branched. Typical oöspores may be formed, or in certain species they may be either rare or entirely suppressed.

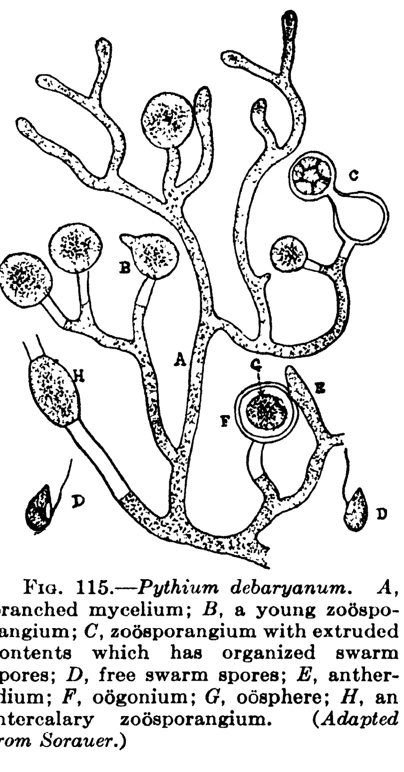

FIG. 115.—*Pythium debaryanum.* *A*, branched mycelium; *B*, a young zoösporangium; *C*, zoösporangium with extruded contents which has organized swarm spores; *D*, free swarm spores; *E*, antheridium; *F*, oögonium; *G*, oösphere; *H*, an intercalary zoösporangium. (*Adapted from Sorauer.*)

The important genera may be briefly characterized.

Pythium.—Swarm sporangia on branches similar to the mycelium, spherical, oval, obpyriform, filamentous, etc. Sporangia germinate by a rupture or by a beak through which the protoplasmic contents are extruded into a thin-walled vesicle, after which they are differentiated into laterally biciliate (uniciliate according to some authorities) swarm spores. Some species produce terminal or intercalary gemmæ or conidia similar to the sporangia, which germinate by swarm spores or after a rest period by a germ hypha. Direct germination is also characteristic of the resting oöspores. (Nematosporangium formerly considered a distinct genus is now merged with Pythium.)

Phytophthora.—Sporangiophores simple or branched, generally emerging through the stomata of the host, but sometimes through epidermal cells. Sporangia at first terminal, but becoming lateral by the further growth of the branch, and thus produced in succession. The germination may be pythiaceous, without the formation of a vesicle, or by the direct formation of an infection hypha. The genus was formerly included with the Peronosporaceæ, but certain species can be scarcely distinguished from Pythium. Phytophthora species differ from the downy mildews in

producing an intracellular growth as well as intercellular, in not being obligate parasites and by the successive development of the sporangia. Oöspores lacking or, when formed, of the typical oömycetous type, or in certain species "amphigenous," that is, with an antheridium in the form of a basal collar. (Phythiacystis, Kawakamia, Belpharospora and Pythiomorpha formerly described as distinct genera are now merged with Phytophthora.)

Trachysphæra.—Sexual stage similar to Phytophthora with amphigenous antheridia; one or more globose echinulate conidia (sporangia) on sterigmata borne on an enlarged vesicle at the tip of the sporangiophore. Germination by germ tube; no swarm spores known.

ALBUGINACEÆ

General Characters.—This family has received the common name of the white rusts because of the abundant production of whitish fruiting pustules which burst through the host epidermis in much the same way as the colored pustules (reddish or black) or sori of the true rusts. The special features which characterize this group are:

1. The production of conidiophores in associated groups, or sori, beneath the host epidermis, which later break through and expose the powdery or dusty mass of spores (conidia—in reality, sporangia).
2. The unlimited production of conidia (sporangia) in chains.
3. The germination of both conidia and oöspores by the production of zoöspores or swarm spores.

The Development of Mycelium and Asexual Reproductive Structures. First infection results in the development of a copious growth of internal, intercellular, non-septate mycelium, the hyphæ showing considerable irregularity in diameter. The mycelium soon organizes the characteristic groups of conidiophores which develop beneath the epidermis, raising it, to make whitish pustules or extended blister-like areas due to the merging of adjacent sori. As soon as the covering epidermis ruptures, the conidia or sporangia are set free and may germinate at once if favorable conditions are offered. These conidia are the so-called "summer spores" and serve for the rapid dissemination of the fungus during the growing season.

The conidiophores are short, basally branched, club-shaped structures and give rise to simple chains of spores. The number of spores produced is indefinite, and they are formed in basipetal succession, that is, the conidiophore forms a cross-wall or septum, cutting off that portion which is to become a spore. This gradually takes on the characteristic form, the conidiophore increases in length, a second spore is cut off and the process continues, resulting in the simple chains of multinucleate conidia, generally separated one from another by short neck-like projections. As conidial production continues, the older, terminal portions of the chains break, setting free the individual conidia.

The conidium does not normally germinate by the production of a hypha or germ tube, but becomes a *zoösporangium.* The protoplasmic contents divide into six or more portions, which emerge through an opening as naked protoplasts or swarm spores, each provided with two unequal lateral cilia or organs of locomotion. They are able to swim about actively, but soon come to rest, absorb their cilia, surround themselves with a cell wall and germinate by the formation of a germ tube. If this germination occurs on a susceptible host, the infection thread will penetrate to form an internal intercellular mycelium.

The Formation of Oöspores.—The same mycelium which has produced *sori* will a little later in the season organize the antheridia and oögonia and produce oöspores which remain within the host tissue. These are the so-called "winter spores," and serve the important function of carrying the fungus over the winter period, as they will not germinate until the following spring.

The oögonia and antheridia are formed on the internal intercellular mycelium, each appearing on separate but adjacent hyphæ. The oögonium is a large globular multinucleate cell, the antheridium a smaller, more or less globular cell. One or more antheridia come to occupy a position close to an oögonium. There are two different types of egg organization within the oögonium. In certain species (*A. candida*) the protoplast becomes differentiated into a peripheral or external zone, the *periplasm,* which contains many nuclei, and a central mass, the *egg cell,* or oöplasm, which contains a single nucleus. In other species of white rusts the central oöplasm remains multinucleate (*A. bliti* and *A. portulacæ*).

The antheridium, which is a multinucleate cell, produces a short, tube-like outgrowth, the *fertilizing tube,* which penetrates the periplasm and comes in contact with the egg. The antheridial or male nuclei are discharged through this tube into the egg cell. In the uninucleate egg the female nucleus fuses with a single male nucleus, and in the multinucleate egg female and male nuclei fuse in pairs, this nuclear union constituting the process of fertilization.

Following fertilization the egg is gradually transformed into a thick-walled oöspore. The periplasm is absorbed, the oöspore wall darkens and thickens and develops characteristic external ridges, reticulations or knobs, while the interior of the oöspore becomes filled with an abundance of reserve food in the form of oily or fatty globules. The fully developed oöspore lies within the old empty oögonial cell.

The oöspores are set free only by the weathering and decay of the host parts in which they were formed. They are resting spores and will not germinate until they have been subjected to winter temperatures. Under favorable spring conditions they germinate by the production of swarm spores. The oöspore wall splits and allows the internal, transparent, sac-like membrane containing the swarm spores to protrude.

This sac finally ruptures and the biciliate swarm spores escape. These swarm spores behave in the same manner as those formed from the conidia and will produce new infections under favorable conditions.

Albugo is the only genus.

PERONOSPORACEÆ

This group has received its common name of the downy mildews from the characteristic production in the typical species of aerial conidiophores in loose white tufts or downy aggregates on the surface of the substratum. The special features which characterize the group are:

1. The production of simple or branched conidiophores which generally emerge in small groups from the stomatal openings.
2. The limited production of conidia (sporangia), a single spore being developed from each branch of the compound sporophores, the maturing being simultaneous, rather than successive as in Phytophthora.
3. The germination of conidia either by the formation of swarm spores or by direct development of infection threads.
4. Oöspores germinate by direct production of a germ tube or infection thread.

The downy mildews generally produce only an internal, intercellular, non-septate mycelium, very similar to that of the white rusts. The aerial conidiophores constitute the most distinguishing feature, and clearly separate the downy mildews from the closely related white rusts. Following an infection there is the development of the characteristic internal mycelium which frequently becomes aggregated in the substomatal chambers. One or several aerial conidiophores grow out through the stomata, and become branched sporophores bearing more or less pear-shaped conidia (sporangia) singly, but never in chains as in the white rusts. The conidia germinate by the formation of an infection thread or germ tube in Peronospora and Bremia species, but in the other genera swarm spores are produced. (*Bremia lactucæ* may also form swarm spores.) These are either set free through a germ pore as fully matured swarm spores, or escape surrounded by a delicate vesicle within which they complete their development. Oöspore formation is essentially similar to that described for the white rusts. A deviation may be noted in Sclerospora, in which the oöspore completely fills the oögonium and becomes closely adherent with the oögonal wall. Oöspores which have been observed to germinate give rise to a germ tube instead of forming swarm spores (except in Sclerospora), or produce a promycelium which forms a single zoösporangium (see Downy Mildew of Grape, page 439).

The following are the most important genera:

Sclerospora.—Mycelium intercellular, with vesicular haustoria; conidial stage inconspicuous in most species; conidiophores stocky and solitary or in groups of two or three; oöspores generally conspicuous,

permanently united with the walls of the oögonia. Oöspore germinating by swarm spores.

Plasmopara.—Sporangiophores slender, tree-like in form, solitary or fasciculate from the stomata of the host, monopodially branched, more or less at right angles, the ultimate branches obtuse. Germination by swarm spores, or the entire protoplasmic mass may escape and then send out a germ tube.

Peronoplasmopara.—Sporangiophores as in Plasmopara, but pseudo-monopodially branched, the branches forming more or less acute angles, with the ultimate portions acute. Germination of sporangia by swarm spores.

Bremia.—Sporangiophores dichotomously branched, the branches terminated by disks or swellings which give rise to short radiately arranged, conidium-bearing branches. Germination of conidia direct or sometimes by swarm spores.

Peronospora.—Conidiophores dichotomously branched, at acute angles, the ultimate branches acute. Germination of conidia direct by lateral germ tubes.

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LATE BLIGHT AND ROT OF THE POTATO

Phytophthora infestans (Mont.) De By.

The late-blight attacks and *kills the tops* of the potato plant and invades the tubers, causing either a *dry* or a *wet rot*. It is undoubtedly the most serious of all the potato diseases when conditions are favorable for its development.

History.—This disease was introduced almost simultaneously into Europe and North America sometime between 1830–1840, the exact date being uncertain. It is, however, certain that the trouble was well established in Ireland, England and on the Continent by 1842, and became widespread by 1845. At about the same time the disease attracted attention in Massachusetts, New York and Pennsylvania. In 1845 the late blight became epiphytotic in both Europe and eastern North America, where it devastated the fields and left famine in its wake. The notable Irish famine of 1845 and 1846 was due largely to the failure of the potato crops, which at that time constituted the staple food of over four million of the people of Ireland. India was not invaded until between 1870 and 1880, and in 1909, Australia, which was long thought to be immune, had the disease recorded in every state. It was also severe in South Africa in 1909.

When the blight first became prevalent but little was known about fungous parasites and most of the early writers attributed it to various causes, such as electricity, some unfavorable atmospheric influence, wet season, wet season combined with

drought and frost, insects, ruptures of the cells, a weakened or impaired constitution or the direct visitation of Providence. Von Martius in 1845 attributed the disease to a fungus, and his opinion was also affirmed by Morren, but their ideas made little headway against the ignorance and superstition of their time. The final acceptance of their views was due to the masterly work of De Bary in 1861–1863. Since 1904 a voluminous literature has accumulated dealing with various phases of etiology and control.

Geographic Distribution.—Although the late blight has become world wide, its occurrence in different countries is limited to a certain extent by climatic factors. In North America the region of greatest severity includes the New England states, New York, New Jersey, Pennsylvania, adjacent Canada and states of corresponding latitude as far west as Iowa and Minnesota. It occurs in less severe form a little

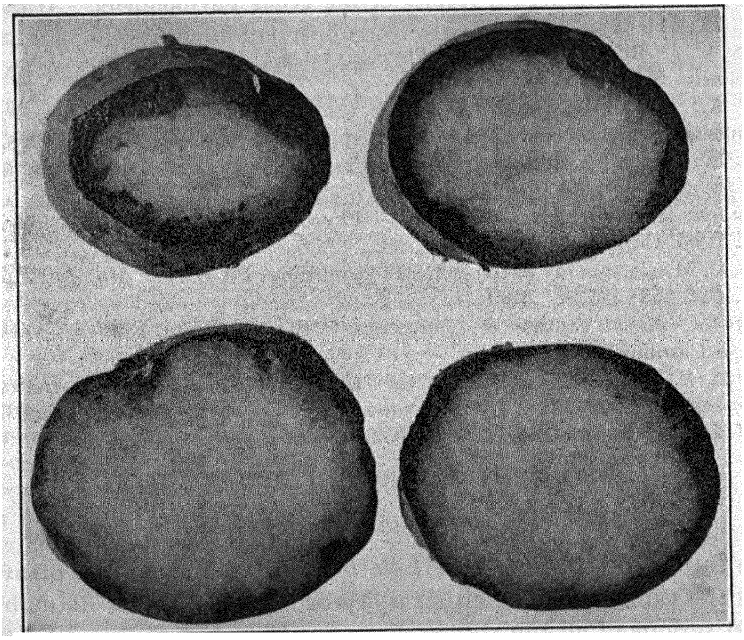

FIG. 116.—Sections through tubers affected with late-blight rot due to *Phytophthora infestans.*

farther westward and in scattered sections of the Atlantic states to Florida. It is also known in Cuba. West of the Rocky Mountains it occurs in several sections in California and Oregon and is of increasing importance in the Puget Sound country.

The disease occupies that portion of Europe which has a climate corresponding to the American regions in which it is prevalent. This includes Russia and other states west to Great Britain and France, in the latter of which it generally reaches great severity. It extends in less severe form as far south as Italy.

The causal fungus is a native of the northern Andes, from which section it was introduced into America and Europe, and recent literature recognizes the potato blight as an important disease of those South American sections which offer favorable conditions.

Symptoms and Effects.—After blossoming time the blight appears on the foliage, where it causes brown, dead spots or extended dead areas more frequently until the leaves are killed. The dead areas appear at

the tips or margins of the leaves and spread downward, the rate of advance depending upon the weather conditions. If moist weather prevails the entire leaf may be killed in from one to four days. If dry weather follows, the infection advances more slowly and the blighted leaves soon curl and shrivel, while under moist conditions, they remain limp and soon decay, often emitting an offensive odor. It is generally the lower leaves that first show the disease but all portions are affected and there are numerous infections in severe cases, primary lesions appearing on petioles and stems as well as upon the leaves.

In dry, clear weather the number of leaf lesions is limited, and the spots remain small and dry up without involving the entire foliage. In warm muggy weather the disease advances very rapidly, the entire tops becoming blackened and wilted, followed by a wet rot involving the stems

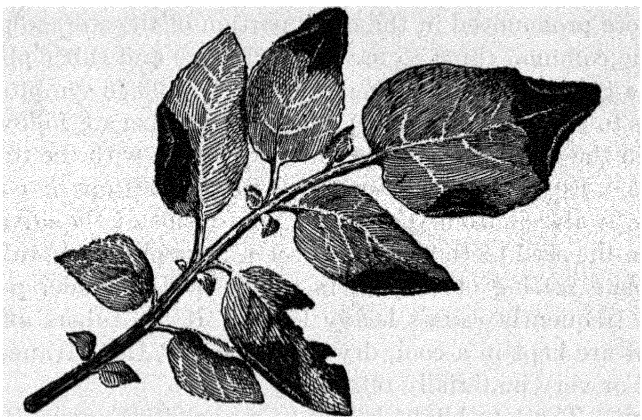

FIG. 117.—Leaf of Irish potato showing terminal and marginal lesions of late blight. (*From Cornell Univ. Agr. Exp. Sta. Bul.* **140.**)

as well as the foliage. The rapidity of spread of the disease is such that the promise of a bountiful crop may be entirely wiped out in a few weeks after the first appearance of the disease. The stem decay does not advance downward into the tubers but separate infections occur.

If blighted leaves are examined while they are still moist and especially after humid conditions have prevailed for a few days, a delicate, whitish or grayish bloom may be observed upon their under surfaces. This bloom is generally most evident where the diseased area borders the unaffected portions. This whitish haze consists of aerial fructifications of the parasite, which have grown out through the leaf pores. In this condition there is an abundant production of spores which may spread the disease to other leaves or other plants. This aerial growth is rather evanescent, and may be scanty or even absent under dry, sunshiny conditions.

The effect of the disease on the tubers is secondary or primary. The early blighting and death of the tops will reduce the size and number of

the tubers, but the primary invasions of the tubers cause more or less rotting, a *dry or a wet rot* resulting according to the conditions which prevail. In heavy, damp soils with suitable temperature, the rot advances through the surface layers and penetrates deeper, first causing a superficial browning and blackening of the tissue. The affected tubers may be completely decayed before harvest as a result of the activity of the blight fungus and secondary invasions by soil fungi and bacteria. This condition constitutes the so-called wet rot. Under less favorable conditions of moisture and temperature the superficial, brown discoloration penetrates only ⅛ to ¼ inch, the affected portions remain relatively firm, while the surface over the invaded tissues becomes slightly sunken and shows a darker color than normal, sometimes becoming purplish black. This *dry rot* may be confined to a few small restricted areas, or large portions of the tuber may be involved. Dry rot may be quite evident at digging time or it may become more pronounced in the early portion of the storage period.

It is the common thing to have both foliage and tuber phases of the disease in a given crop but in certain cases the foliage symptoms may be so slight as to attract little attention, while the tuber rot follows in severe form; again the foliage attack may be very severe with the tuber rot less in evidence. It has also been shown that tuber infections may result when the disease is absent from the foliage, as a result of the advance of the decay from the seed piece along the stolon (Murphy and McKay, 1927). The complete rotting of the tubers in the field or under poor storage conditions frequently causes heavy losses. If the tubers affected with the dry rot are kept in a cool, dry storage cellar, the advance of the rot is checked or very materially retarded.

Etiology.—The late blight is due to a parasitic fungus, *Phytophthora infestans.* This fungus was described by Montagne in 1845 as *Botrytis infestans.* For some time following this it was treated as a species of Peronospora, but in 1875 De Bary proposed the new genus Phytophthora on account of the peculiar mode of production of conidia. The fungus was first considered only as a saprophyte and an accompaniment of certain physiological disturbances. Several early workers had demonstrated the parasitism by inoculations. Speerschneider in 1857 first showed that the spores from the leaves could carry the disease to the tubers, but the most complete and thorough demonstration of the parasitic nature of the trouble was offered by the masterly work of De Bary. The continued studies in later years by various pathologists have materially added to our knowledge of the causal organism and its exact relation to the disease.

The fungus develops an internal, non-septate mycelium of large thin-walled hyphæ which traverse the intercellular spaces of affected parts, leaves or tubers, and send single or double, club-shaped haustoria, that are sometimes hooked or even spirally twisted into the cells (Szymanek,

1927). Slender aerial hyphæ, the *conidiophores*, grow out through the stomata in groups of one to five. The conidiophores are sparsely branched, relatively thick-walled, show cross-partitions and the side branches bear bulbous enlargements at intervals. The ovoid conidia

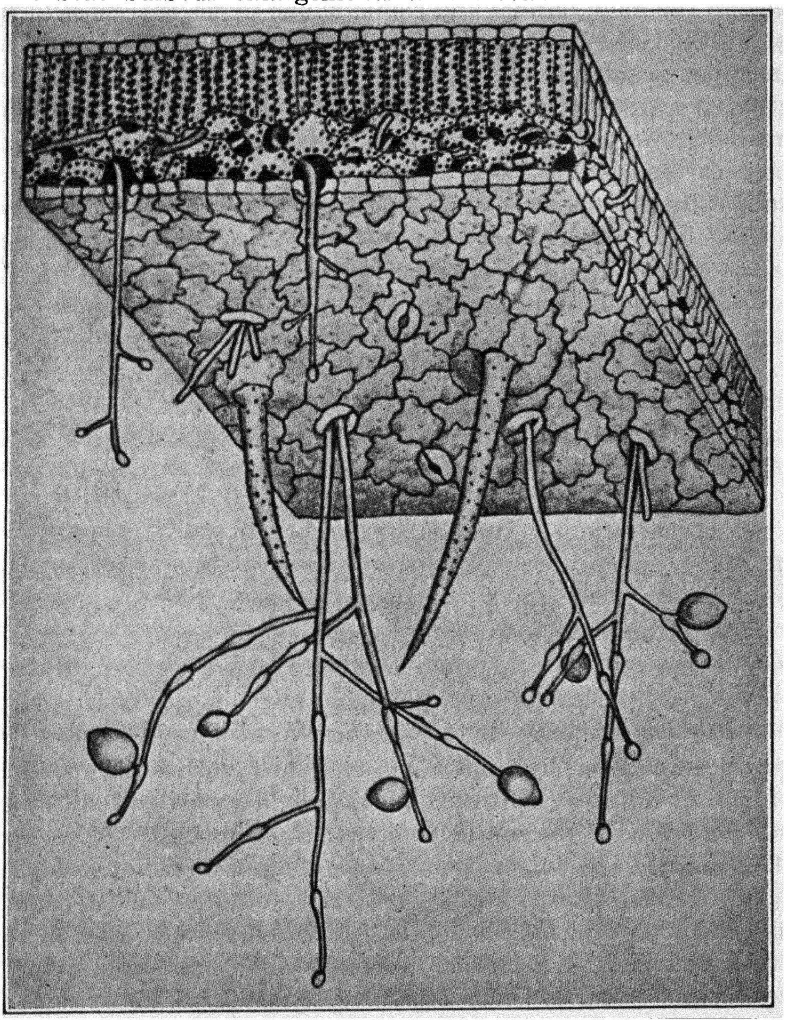

FIG. 118.—Diagrammatic representation of a square cut from a potato leaf infes... with *Phytophthora infestans*, showing the fungus emerging through the stomata and the successive stages in the development of conidiophores and conidia. (*Redrawn from Vt. Bul.* **168**, *by Jones, Giddings and Lutman.*)

are at first terminal, later becoming lateral as the parent branch continues its growth. The enlargements mark the places where conidia were attached. The conidia are multinucleate (7 to 30), ovoid or lemon-shaped, 22 to 32 by 16 to 24μ, and provided with a short stalk and an apiculate tip. Conidia may rarely be produced on internal branches.

The conidia germinate either directly by sending out a germ tube or infection thread or indirectly by the formation of swarm spores. The swarm-spore formation and behavior are essentially similar to that described for Albugo. This latter method of spore germination must be considered the more normal and common one under natural field conditions, but the type of germination is influenced by temperature, moisture and medium or substratum.

Ever since 1847 when Unger recognized that the late-blight fungus was really a downy mildew, there have been speculations and disputes as to the occurrence of oöspores. Oöspore-like bodies have been found in artificial cultures by a number of different investigators (Jones, Clinton,

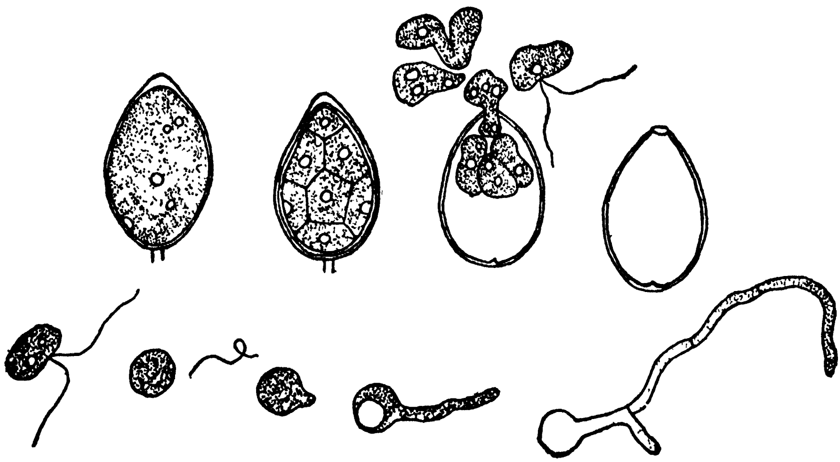

FIG. 119.—Stages in the germination of a conidium (zoösporangium) of *Phytophthora infestans*, and the germination of a swarm spore to form an infection thread. (*After Ward.*)

Pethybridge and Murphy), but true sex organs and resting spores have been found in cultures (DeBruyn, 1926) and on plants and tubers under natural conditions in the soil (Murphy, 1927; Szymanek, 1927).

The conidia are mainly responsible for the new infections which may take place through any part of the epidermis of leaves and stems, either through stomata or the unbroken cells. The germs tubes are even able to penetrate the surface of unmatured tubers, probably entering through the lenticels. Infection may occur through either upper or under surfaces of the leaves, but the lower surface seems to be more susceptible. The conidia are supposed to be short-lived, retaining their viability for two or three weeks only, but recently conidia produced by infected plants which failed to reach the surface were found to be viable 57 days after planting. Since they cannot survive the winter period, the method of persistence of the fungus from one season to another has been a much mooted question.

At least six theories have been advanced to explain the yearly occurrence of the blight: (1) the persistence of the mycelium in the soil; (2) a

perennial mycelium within the affected tubers; (3) the production of oöspores or resting spores which live over in the soil; (4) the presence of a latent mycelium or "mycoplasm" within the potato tissues; (5) the fruiting of the fungus on the parent tuber in the soil; (6) the development of sclerotia-like bodies.

The first two theories have seemed the most probable, but the experiments of Stewart are opposed to the overwintering of the blight in the soil, while the recent finding of oöspores in nature (Murphy, 1927) lends support to the possibility of overwintering in that stage. The work of Melhus (1915), Salmon and Ware (1926), and Murphy and McKay (1927) has established the findings of earlier workers that the disease frequently originates from the use of infected seed. In such cases the mycelium from the parent tuber grows upward in the stems and sporulates on the small dwarfed shoots of a hill. Infections upon new foliage start from the spores produced by these primary infections and the disease soon gains headway. Since infected seed pieces producing shoots that fail to reach the surface have been observed to produce mycelium and conidia for a distance of 2 centimeters from the shoot, it is conceivable that some of these conidia may be brought to the surface by cultivation or insects and thus start new infections (Murphy and McKay, 1927).

Fields which contain no primary infections from diseased tubers may become infected from neighboring fields, since the spores may be spread by the wind and by leaf-eating insects.

The general infection of tubers accompanying an attack of blight is by means of spores which were produced on the blighted tops and are subsequently washed into the soil. Contact of tubers with freshly blighted tops at digging time is also responsible for infections, while there may be some spread from infected tubers to sound ones either in the soil or in storage, if sufficient moisture is present.

Loss from Blight.—The loss from blight is due to the reduction in yield caused by the premature death of the vines, and the accompanying rot which may destroy the tubers, or lower their market value by partial dry rot or by impairing their keeping qualities. At various times in certain regions the disease has caused almost a complete crop failure, while in the eastern United States losses of 50 to 75 per cent have been repeatedly noted. The amount of injury from the disease may be inferred when it is noted that in regions where the disease is prevalent its efficient control has resulted in yield increases of 40 to 233 bushels per acre.

Ever since the great epiphytotic of 1845 and 1846 there have been repeated occurrences of the disease in severe form. The amount of loss has been high in unprotected fields, as illustrated by the estimate for Vermont for 1901, where there was 65 to 95 per cent of rot in the tubers. The loss in New York in 1903 was estimated to be 50 bushels per acre on the average or 20,000,000 bushels for the state.

Climatic Relations.—Excessive humidity coupled with the suitable temperatures are the principal predisposing factors. Where the mean temperature exceeds 77°F. the disease is reported to be unknown. Both germination of conidia and subsequent infection are influenced by temperature. According to Melhus, the optimum for germination lies between 10 to 13°C. (12 to 14°C., Vowinckel, 1926). For direct and swarm-spore germination he makes the following report:

	Minimum, degrees Centigrade	Optimum, degrees Centigrade	Maximum, degrees Centigrade
Swarm-spore germination	2–3	12–13	24–25
Direct germination	10–13	24	30

The living mycelium in the tuber is killed by exposure to 40°C. for 4 hours or at 30°C. for 65 hours according to European investigators, but in Australian tests it required 4 hours at 49°C. Butler reports that the mycelium in either tubers or pure cultures soon dies out at the high laboratory temperatures during hot weather (30 to 35°C.). Jones gives 16 to 18°C. as the optimum temperature for the growth of the mycelium. The optimum is given by Vowinckel (1926) as 19 to 22°C., with the minimum 4.6°C. and the maximum 27°C., while fructification is confined to a narrower range of temperatures (8.7 to 26°C.). It is undoubtedly the temperature factor which has excluded the disease from the Great Plains and the southwestern United States, since the disease is checked if the mean daily temperature is above 75°F. for a few days. Epiphytotics are likely to occur when unusually cool weather, combined with abundant precipitation, prevails at time the conidia are being produced. A warm, humid period followed by a drop to 60°F. is very liable to initiate an attack. This effect of temperature is due to the increased germination of the conidia and not to any modification of the susceptibility of the host. Four important conditions for the development of late blight in severe form have been suggested: (1) night temperatures below the dew point for at least 4 hours; (2) a minimum temperature of 10°C. or slightly above; (3) mean cloudiness of 0.8 or more the next day; and (4) rainfall during the next 24 hours of at least 0.1 millimeter (Van Everdingen, 1926). It is of interest to note in this connection that Löhnis (1925) reported that there is no correlation between frequency and amount of rainfall, vapor pressure, relative humidity and temperature and, the occurrence of an epidemic.

Host Relations and Varietal Resistance.—The late-blight fungus affects various other species of the nightshade family (Solanaceæ). It may be destructive to tomatoes, both in the field and under glass, causing

a blight and fruit rot resulting in heavy losses in transit to market (Giddings and Berg, 1919; Ramsey and Bailey, 1931). It is sometimes destructive to peppers and eggplants and occurs on numerous other less important species. It is of interest to note that it has been found on the "edible fruits of *Solanum muricatum* at the equator, on *Solanum caripease* at Quito, and on Petunia hybrids at Upsala." It has also been described as affecting two species of figworts (Scrophulariaceæ): *Schizanthus grahami* and *Anthocercis viscosa.*

Jones (1912) has shown that different varieties of potatoes show varying degrees of resistance to blight. Resistance of foliage manifests itself by fewer infections and slower progress of the mycelium through the mesophyll after infection has taken place. There is also a similar rate of variation in the growth of mycelium through tuber tissue. In more recent work it is claimed that there is no positive correlation between susceptibility of foliage and susceptibility of tubers (Vowinckel, 1926). Based on growth in tubers the following groups were recognized by Jones in testing 76 varieties: (1) highly resistant; (2) moderately resistant; (3) intermediate; (4) moderately susceptible; and (5) very susceptible. It is of particular significance to note that European varieties predominated in the highly resistant group, while the larger number of the very susceptible varieties were American.

It is generally agreed that resistance is in part definitely varietal and based on the presence of some substance or substances in the tissues which retard or inhibit the growth of the parasite, but it is also recognized that susceptibility changes during the period of growth and may be greatly modified by external conditions. It is claimed that susceptibility varies with the water-nitrogen ratio, increased nitrogen affording increased resistance (Collins, 1925), the water content generally being highest at the time of the principal seasonal infection. Early varieties are stated to be more susceptible because of higher water content of the leaves, while very late varieties having a lower water content are most resistant. Plants grown in dry soil are reported to be more resistant than those grown in moister soil (De Bruyn, 1926). Shortage of potash in the soil is reported to lessen infection, not by a host response but by retardation of sporulation thus reducing the quantity of inoculum (Vowinckel, 1926). Susceptibility to infection increases with age; hence early-maturing varieties are attacked before late varieties (Müller, 1931).

Infection of tubers takes place through either lenticels or eyes as the infection courts, and lenticel character may be modified by environmental conditions (Löhnis, 1925).[1] Lenticels of Eigenheimers grown on clay are generally formed of parenchyma cells with unsuberized walls, while those from sandy soils are suberized, the latter giving resistance and the former susceptibility. It is further stated that sporangia falling on clay

[1] *Rev. App. Myc.* **4**: 761–763. 1925.

soil remain viable longer than on sand. A close correlation between resistance and the thickness of the cell walls of parenchyma and of the middle lamellæ of tuber cells has been reported (Szymanek, 1927).

Some progress has been made in the breeding for resistance, using the more resistant but undesirable varieties crossed with varieties showing desirable qualities. Hybrids between Ekishiraza, a highly resistant Japanese variety, and Irish Cobbler and others have yielded 46 families with resistance and desirable qualities (Reddick, 1928). In this work Müller (1928) has investigated over 700 varieties of varying origin. One of the important aims in the breeding has been to secure later maturing (8 to 14 days). It is reported that these resistant hybrids in Germany have given higher yields in epidemic seasons than the older, susceptible varieties and that they are resistant to all biological forms of the parasite. Crosses between South American and cultivated varieties (Müller, 1930) have yielded six immune hybrids.

Control.—The late blight can be effectively controlled by spraying, and excellent results have been obtained by dusting, but attention should also be given to other measures:

1. *The Selection of Seed.*—Tubers from infected fields should be discarded for seed purposes or very carefully inspected at cutting time and all suspicious tubers rejected. Since the fungus is internal in the seed tubers, the chemical steeps effective for other seed-borne diseases are without value. Disinfection by dry heat which is effective (104°F. for 4 hours) has never proved practical.

2. *Spraying.*—When this is necessary, Bordeaux should be used, and the first applications should be made when the plants are 6 to 8 inches high. For the earlier sprayings the 4-4-50 formula should be used, while the 6-6-50 strength is recommended for later applications. Two or three sprayings may suffice, or they may follow at intervals of 10 to 14 days throughout the balance of the growing season. During epidemics it is sometimes necessary to shorten the intervals and spray as often as once a week. Two to six applications are necessary.

Copper-arsenic dust was first used for late blight of potatoes by Sanders in Nova Scotia in 1918 and increased yields were reported over those obtained from fields protected by the standard Bordeaux applications. Very similar results were reported later by Whetzel, in New York, but in trials in other widely separated states Sanders' copper-lime dust has not give a protection equal to that of Bordeaux (Stewart, 1924). Sanders' dust for potatoes is a mixture of hydrated lime, finely ground, partially dehydrated copper sulphate and calcium arsenate. As a result of trials extending through 4 years, Stewart (1924) states:

> In all four years the differences in yield were decidedly in favor of the spray. Fairly severe attacks of three of the principal foliage troubles—early blight, late blight, and hopperburn—were involved in the experiments. No one of the

three was controlled in as satisfactory a manner by the dust, even though the quantity of dust used was considerably greater than that recommended by advocates of the dust method.

Poor control by dusting is reported by Wallace (1925) as a result of 7 years' tests. Copper-lime dust has given better protection than any other dusts. The difference in yields of sprayed and dusted plots is not great, hence growers may prefer to practice dusting in certain cases because of its convenience. These results are in general agreement with later experiences (Neuweiler, 1926; Muskett, 1929; Schander and Staar, 1930), although Bonde *et al.* (1929) secured slightly higher yields from copper-lime dust.

Spraying or dusting is a protection against infection and is too late if delayed until the blight is evident upon the foliage. A grower must decide whether the disease is sufficiently severe in his region to justify the expense of spraying or dusting. In regions of frequent occurrence and great severity of the disease spraying has become a routine practice. In this connection it is of interest to note that in Holland an arrangement has been made for the Dutch Meterological Institute and special observatories in the potato-growing districts to issue warnings whenever conditions are such as to justify the expectation of an outbreak of late blight (Van Poeteren, 1928).

3. *Storage at Low Temperatures.*—The spread of the dry rot in affected tubers can be very greatly retarded by storage in a cool, dry cellar. Moisture and high temperatures favor the spread of the rot. At storage temperatures of 40°F. or under, the advance of the rot is very slow.

4. *Miscellaneous Practices.*—Attention may be directed to a number of practices which influence the development of the rot in affected fields. The crop from such fields should not be harvested until a week or more after the death of the tops. There would be no objection to a longer delay unless the season is wet and the soil heavy. High-ridge culture, which will bury the tubers 4 to 5 inches, is said to lessen infection. Spraying the surface of the soil with Bordeaux or copper sulphate alone will lessen the amount of rot even though the foliage has not been protected. In late attacks of blight when spraying has not been practiced, the cutting and the destruction of the tops have been productive of good results.

Profits from Spraying.—It has been definitely demonstrated that spraying for blight pays in those regions where the disease is an important factor. Two notable illustrations may be cited. The average gain per acre for 20 years (1891–1910) in Vermont was 105 bushels as a result of spraying two or three times with Bordeaux mixture. The yearly gains varied from 32 to 224 bushels per acre. In New York the average gain per acre for 10 years (1902–1911) was 97.5 bushels, while the yearly gains varied from 39 to 233 bushels. The New York experiments showed that potatoes could be satisfactorily protected for a cost of $4.74 per acre.

More recently in Germany Neuweiler (1926) has reported increased yields from spraying in 80 out of 97 tests.

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THE WHITE RUST OF CRUCIFERS

Albugo candida (Pers.) Kuntze

This is the most common species of the white rusts which attacks foliage, stems, flowers or fruits of various cruciferous plants throughout the world, causing hypertrophy, deforming of parts or blighting of the affected structures.

Symptoms and Effects.—Early in the progress of the disease, the trouble becomes evident by the appearance of the prominent white pustules or blisters, the *sori*, which may appear on any part of the host except the roots. These pustules vary in size and shape and often become confluent to form more extended patches. When young they appear like unbroken blisters, but later break and become powdery.

In many cases the affected leaves are not greatly modified, the spore pustules appearing on one surface only, while in other cases they may be more numerous and appear on both surfaces. There are all gradations from a few localized infections to complete invasion, and the amount of injury will depend upon the degree of infection. On some hosts the

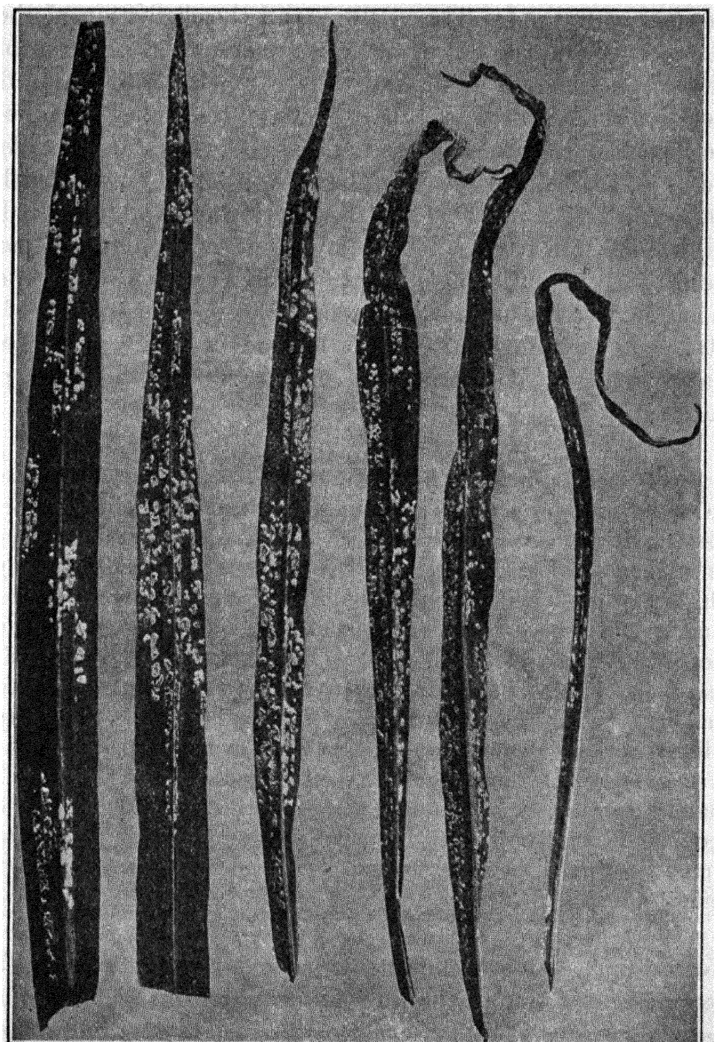

FIG. 120.—Habit sketch of leaves of salsify affected with white rust (*Albugo tragopogonis*), showing sori similar in external appearance to those of *A. candida.*

leaves from infected stems may be "thickened, fleshy, pallid and distorted or inrolled." In severe infections which involve the stems, there may be more or less reduction in the size of the leaves.

The stems which are invaded sometimes show but little change of form, but frequently there are localized or extended swellings. The stem

enlargements may be slight or very pronounced and up to several times the normal diameter, and exhibit sharp bends, turns or even complete spirals. In some cases there may be a proliferation from lateral buds that are normally dormant, resulting in a bushy growth.

The entire inflorescence may be invaded or various flowers or flower parts may be deformed. The swelling and the distortion of the axis of the inflorescence and the flower pedicels are frequently more pronounced than those of stems and in the radish may reach ten to fifteen times the diameter of normal structures. In other cases the axis of the inflorescence and the flower stalks are unaltered and the flowers alone invaded, while severe axis invasions may result in the prevention of flower development. When affected flowers do develop, they may show various discolorations and malformation of parts. The following general modifications may be possible: (1) All or only single floral organs may be swollen and fleshy; (2) the affected organs may be green or violet in color instead of normal; (3) petals and stamens may persist instead of falling; (4) the normal cyclic arrangements of flower parts may revert to the more primitive strobilate type.

The sepals may be immensely increased in size and thickness, often deeply concave on the inner surface, and sometimes provided with cylindrical or flattened appendages near the base. The petals are also enlarged, vary much in shape and sometimes have sepaloid characters. The stamens are usually greatly altered, being thickened into a club-shaped body in which the anthers are represented by an oblong, grooved, green mass, or transformed wholly, or the anthers only, into small, leaf-like structures. In some hosts, the stamens are less affected and may even bear pollen in a portion of the pollen sac, the rest being sterile; occasionally supplementary pollen sacs are formed. Of more interest is the appearance of stigma-like structures at the tip and rudimentary ovules on the margins of certain altered stamens, which thus come to have the characters of carpels. The pistil itself may be enormously swollen into a conical, thick-walled sac, or transformed into a couple of carpellary leaves, borne on a common stalk. In the former case ovules may be formed, but they remain sterile and dehiscence of the fruit does not occur (Butler, 1918).

Affected organs may sometimes appear darkened towards the end of the growing season due to the internal development of large numbers of brown-walled spores. This internal darkening is sometimes more evident with examination by transmitted light and is very noticeable when such affected organs are cut across.

When the white rust occurs alone it may cause little or no injury or it may prove more serious. Its most serious effects are sometimes noted when it is associated with the downy mildew (*Peronospora parasitica*). Seedlings may be killed outright under favorable conditions, while plants less severely affected or invaded later in their development may be

dwarfed. The arrest of flower development or the malformation of flowers results in sterility, and so may be injurious to seed crops.

Etiology and the Causal Organism.—The white rust of Crucifers was first described in 1791 by Persoon as an Æcidium and later in 1801 as Uredo. The early use of these two generic names may perhaps explain

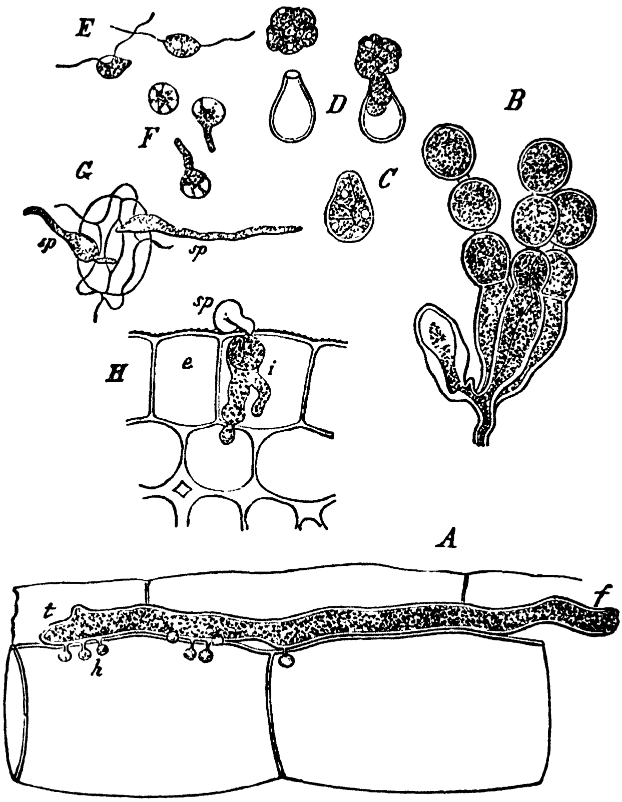

FIG. 121.—*Albugo candida.* *A*, a hypha with growing tip *t* and haustoria *h* between the pith cells of *Lepidium sativum;* *B*, a group of conidiophores and conidia; *C–E*, stages in the formation of swarm spores from conidia; *F*, germination of a swarm spore that has come to rest; *G*, germinating swarm spores about to send infection hyphæ into a stoma; *H*, an infection hypha of *Phytophthora infestans* penetrating the epidermal wall of potato stem. (*After De Bary.*)

the origin of the common name. The genus was described as Albugo in 1820 and as Cystopus in 1848. In all of the earlier American literature the fungus appears as *Cystopus candidus* Lev., but more recently as *Albugo candida* (Pers.) Kuntze.

The development of the internal mycelium, conidiophores and conidia, and oöspores has been described in the general consideration of white rusts. The conidiophores are clavate, thick-walled, 35 to 40 by 15 to 17μ; the wall of the terminal conidium of a chain is thickened on its external side and the conidium is not capable of germinating, but all others in the

chain are set free by the solution of the jointed necks. The separated conidia, which are globular, hyaline, 15 to 18μ, with uniform, thin cell walls, are easily disseminated during dry weather by air currents. These conidia or sporangia may germinate at once if they are afforded favorable conditions of moisture and temperature, producing mature swarm spores in from 2 to 10 hours after immersion in water. The conidia are relatively

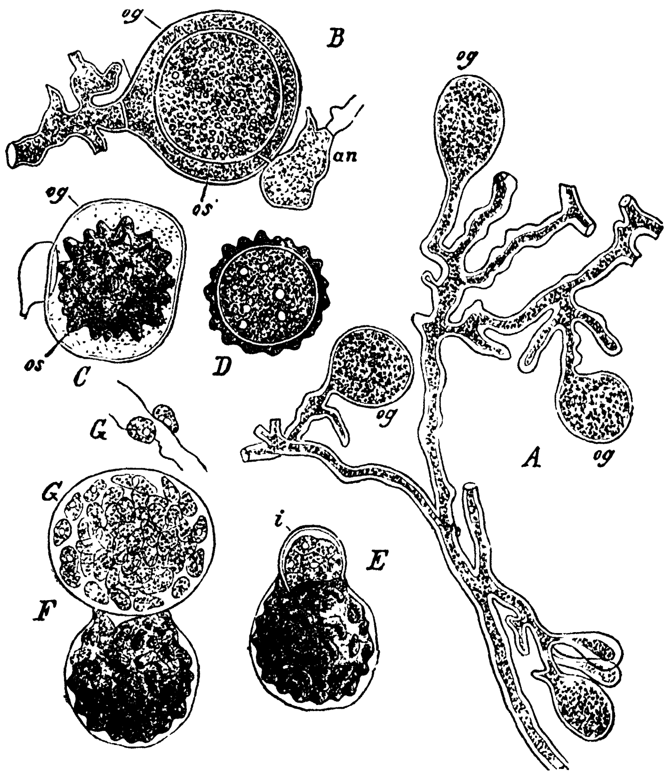

FIG. 122.—*Albugo candida.* *A*, mycelium with young oögonia *og*; *B*, oögonium *og* with the egg cell or oösphere *os* and the antheridium *an*; *C*, a mature oögonium with a fully developed oöspore *os*; *D*, optical section of oöspore shown in *C*; *E–G*, successive stages in the germination of an oöspore with the formation of swarm spores. (*After De Bary.*)

short lived, their period of viability being limited to about 6 weeks after maturity. In germinating, the conidium (sporangium) shows a segmentation of the contents into four to eight polyhedral masses, which separate and escape one by one to the outside or in adherent groups or the whole mass may be discharged into a bladder-like structure. The swarm spores take on their typical form of ovate to kidney-shaped bodies, two unequal cilia appear from the flattened or concave side, and they swim away from the mother cell. After a period of activity they come to rest, absorb the cilia, form a surrounding cell wall and germinate by a germ tube or infection thread entering the host through stomata. It was

formerly believed that first infection took place through cotyledons or seed leaves only, but Melhus has recently shown that various parts of older plants may become infected if suitable conditions are afforded. An incubation period of 7 to 10 days is required, the period being 1 to 10 days longer for the less susceptible species (Huira, 1930).

Oöspores are not known on some hosts, but in many cases they are produced in abundance in the hypertrophied parts. They are rare in affected leaves except in a few hosts. Mature oöspores are globular, 40 to 55μ in diameter and show thick, brown walls provided with low, blunt ridges often confluent and irregularly branched. The oöspores are resting structures and do not germinate at once, but only after some months. Those which have been left in plant débris from the previous

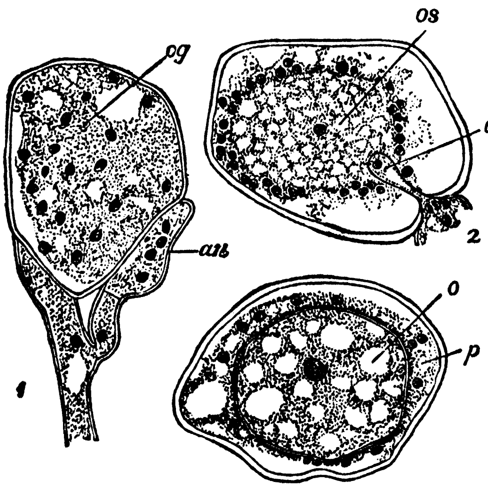

FIG. 123.—Fertilization in *Peronospora parasitica* (1), and *Albugo candida* (2, 3); *og*, young multinucleate oögonium; *an*, antheridium; *os*, uninucleate oösphere or egg; *a*, fertilizing tube of the antheridium which introduces the male nucleus; *o*, fertilized egg cell surrounded by the periplasm. (*After Wager.*)

season's crop or have been set free by the disintegration of affected plant structures probably constitute a very important source of the first spring infections. The germination of the oöspores is typical for the group. Many crucifers produce rosettes which live over winter, and it is possible that these may be infected in the fall and thus carry the fungus over the winter as a dormant mycelium.

There seem to be two types of infection: first, a general or systemic, in which the whole plant is affected, resulting in a stunted growth and the appearance of the spore pustules on all parts; second, a local infection, in which single leaves, stems or flower parts are directly invaded.

Predisposing Factors.—The proper temperature is the most important factor influencing the appearance of the disease. Temperature conditions affect not only the germination of the spores but also the apparent sus-

ceptibility of the host. Conidia germinate better at low than at high temperatures. The definite optimum has not been determined but it is close to 10°C. The minimum temperature is very close to freezing, while the maximum is about 25°C. Spore germination proceeds normally during the cool spring season when there is also an abundance of moisture, but in many regions the summer temperatures would be sufficiently high to check or very greatly lessen germination. The chilling of the host is an equally important factor in inducing infection. According to Melhus, 95 per cent of seedlings chilled became infected, while the controls not chilled generally showed less than 5 per cent and never more than 15 per cent infection. It has been pointed out that the fall in temperature, which leads to the deposit of the dew, provides the stimulus for spore germination, probably increases the susceptibility of the host and at the same time furnishes the medium in which the swarm spores develop. The temperature relations offer the very evident explanation for the greatest development of the white rusts during the cool periods of early spring.

Host Relations.—The white rust of crucifers is found on many species of the mustard family throughout the world, both wild and economic plants being affected. The fungus is not confined to the Cruciferæ, but occurs on various species of Capparidaceæ in Europe and in India.

The most important cultivated hosts for America are as follows:

Cabbage (*Brassica oleroacea* L.)
Cauliflower (*Brassica oleracea* var. *botrytis* L.)
Cress (*Lepidium sativum* L.)
Mustard (*Brassica nigra* K.)
Mustard (*Brassica alba* Rabenh.)
Horseradish (*Roripa armoracia* (L.) Hitch.)
Radish (*Raphanus sativus* L.)
Rutabaga (*Brassica campestris* L.)
Turnip (*Brassica napus* L.)
Watercress (*Roripa nasturtium* (L.) Hitch.)
Wallflower (*Cheiranthus cheiri* L.)
Stocks (*Matthiola incana.*)

In addition to the above, Wilson lists 40 wild hosts from 21 different genera. The most common weed hosts are shepherd's purse (*Bursa bursa-pastoris*), pepper grass (*Lepidium virginicum*) and *Sisymbrium officinale.*

There is some evidence to show the existence of specialized races or biological species, although further evidence is necessary to determine their limits. According to Melhus, the form on the common radish passed readily to other varieties of *Raphanus sativus* and also to *R. caudatus*, and less frequently to white mustard (*Brassica alba*) and cabbage (*B. oleracea*), but failed entirely when inoculated on 10 other species.

Another observer has reported the strain from turnips capable of infecting the cabbage and its derivatives. *Arabis alpina*, a weed, has furnished spores which infected nine other species belonging to six different genera, but this strain failed on radish, mustard and cabbage. Recent studies in Japan have revealed at least three physiological strains: one from radish affecting all radishes but no other species; another on Chinese mustard, (*Brassica juncea*); and a third on *Brassica campestris chinensis* (*Hiura*, 1930). The second and third strains were able to infect different groups of Brassica species.

Prevention or Control.—The white rust is not generally sufficiently severe to justify expensive control measures. Attention should be called to certain practices which will be of value: (1) crop rotation to prevent the growth of susceptible crops in ground filled with the overwintering spores from a previous infected crop; (2) clean culture to keep down all cruciferous weeds; (3) the destruction of infected crop refuse by burning to prevent carrying oöspores over the winter. Spraying is recommended only in the case of very severe attacks.

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DOWNY MILDEW OF GRAPE

Plasmopara viticola (B. & C.) Berl. & De T.

The downy mildew of the grape affects leaves and young stems, causing a *spotting or blight*, and may also invade the berries, causing *rot* or shattering. As a grape disease, it probably stands next in importance to the black rot.

History.—This disease was first observed in America on wild grapes in 1834. Early writers recorded the occurrence of "mildews" and "blights" of vines, but these troubles were not at first associated with the presence of a parasite but attributed to various causes, such as atmospheric disturbances, or even some visitation of Providence. The disease may be considered endemic in the eastern United States, from

which region it spread to France, perhaps sometime previous to 1874, the first severe infestation being in 1879. The spread to other parts of Europe followed. It is interesting to note that the disease did not reach South Africa until 1907, that the first severe outbreak in Australia occurred in northeast Victoria in 1917, but did not reach New Zealand until 1926. The parasite was first described by Berkeley and Curtis in 1848, but the fungous origin of the trouble was recognized by Schweintz at an earlier date (1834). De Bary studied the fungus very carefully in 1863, Farlow in 1876 and Berlese and De Toni in 1888, when it was assigned to the present genus. On account of its importance and severity, the various aspects of the disease have been treated by many different writers in a voluminous literature.

Geographic Distribution.—The disease is present to a greater or less extent in nearly all parts of the world where grapes are cultivated. The important vineyards of France have suffered severely because of the general use of the very susceptible *Vitis vinifera* varieties, coupled with the very favorable climatic conditions. Severe epiphytotics have been recorded for 1900, 1910, 1915 and 1927 in France. The mildew visitations are less intense in southern France than in the moister center and north. In northern Africa (Algeria) the dry inland regions suffer little, while in the coastal belt the disease causes trouble, although it is much less severe there than in France. The disease first appeared in Australia and South Africa during or following periods of rather exceptional weather conditions. The fungus is present in South America and it is claimed that the summer stage is present the year round in Brazil.

In the United States the mildew is prevalent east of the Rocky Mountains, and reaches its greatest severity in the northern Mississippi Valley and states to the eastward, especially the Middle Atlantic states. High temperatures and dry conditions have been unfavorable for the disease in the South, the disease being very rare and of no economic importance in the arid Southwest. The downy mildew has never been found as a disease of cultivated vines in California, although a single collection on a wild species has been reported. The exclusion of the disease from the grape districts of California and other regions west of the Rocky Mountains seems to be due largely to unfavorable climatic conditions.

Symptoms and Effects.—The downy mildew on the leaves first shows upon the upper surface in the form of pale-yellow spots of variable size and form, frequently more or less circular, which merge into the surrounding green tissue without any distinct line of demarcation. In the early stages these spots appear more transparent than the normal leaf tissue, this character being especially noticeable if affected leaves are held up to the light. This peculiarity has suggested the French name of "oil spots." These spots show less distinctly on the lower surface at first, owing to the covering of hairs, but under favorable atmospheric conditions the lower surface of each spot is soon occupied by a conspicuous aerial growth of the fungus in the form of a downy, milk-white coating, which suggests the common name "downy mildew." As the affected spots become older, the leaf tissue may be gradually killed, the color changing from yellow to a brown or dead-leaf color, and in this condition the spots are more evident upon both surfaces. Under dry conditions the aerial downy growth on the under surface of the leaves may be absent or scanty, and this condition prevails on the older spots which have changed to the brown color. The leaf spots may be few in number or they may be so numerous as to coalesce and involve nearly the entire leaf or extended

portions. In the more resistant varieties the leaf spots do not become so conspicuous as in the very susceptible varieties, the invaded areas being mottled or punctate brown instead of uniformly colored.

Another vine trouble known as erinose, due to a mite, shows symptoms which sometimes suggest downy mildew. Erinose spots are always convex above, while mildew spots are flat. While the down or felt of erinose is white at first as in mildew, it soon changes to a rusty brown.

FIG. 124.—Under side of a grape leaf showing groups of conidiophores of *Plasmopara viticola.*

The young canes, leaf stalks and tendrils are also subject to attack. The affected portions show a water-soaked appearance at first, later turning yellowish green, but finally becoming brown. The external development of the mildew may appear on these structures also, or it may be entirely suppressed.

In older lesions the dead tissues shrivel, causing a depression. The effect of the cane lesions will depend upon their number and extent. In extreme cases the young shoots may be dwarfed, twisted or deformed and the leaves remain small, while in the most severe infections the cane may die. Lesions on the leaf stalks may be instrumental in causing the shedding of the leaf.

Flowers and fruits may be invaded and blighting or rotting result. Fruits may be attacked when young or when approaching maturity. If the berries are attacked when young, that is, when about ¼ inch or less in diameter, further growth is checked, the gray superficial growth of the fungus appears, the berry darkens and finally dries up. In berries attacked when nearly full grown, but before color appears, the fungus only rarely appears on the surface, but the infected berries darken due to the death of the constituent cells. This change progresses slowly, dark patches appearing at separated points, the skin becomes withered and wrinkled and finally the whole berry becomes shrunken and dark brown. The condition on the younger fruits with the copious growth of the mildew has sometimes been designated as *gray mold*, while the characteristic changes of the older fruits have suggested the name of *brown rot*.

The effects of the disease may be briefly summarized:

1. The normal physiological activities of the leaves are interfered with, the amount of injury depending upon the area of leaf surface involved. Vines thus stripped of their leaves early in the season are not able to ripen their fruit normally. Fruits from such vines contain less juice and have a lower sugar content. The crop of the following season is also seriously menaced, since the usual accumulation of reserve food cannot take place.

2. Young canes may be dwarfed and the leaves remain small, or the cane together with its leaves may die.

3. Fruit attacks cause either mummification or rotting, and shelling or dropping of the diseased berries is the common result. The shelling may be almost complete or result only in poorly filled bunches. It is estimated that there was a loss of 70 per cent in France in the epiphytotic of 1915.

Etiology.—The downy mildew is caused by *Plasmopara viticola* (B. & C.) Berl. & De T. Berkeley and Curtis described the grape mildew in 1848 as a new species, *Botrytis viticola*, but De Bary in 1863 determined its true relationship and named it *Peronospora viticola*. It was not until 1888 that Berlese and De Toni redescribed the fungus under its present name.

The characteristic cœnocytic mycelium develops within the intercellular spaces of affected tissues, forming thin-walled hyphæ of very irregular diameter, varying from 1 to even 40 to 60μ, the greatest regularity appearing in the compact tissues, while the greatest irregularity and size may be found in loose leaf tissue with prominent intercellular spaces. The hyphæ do not penetrate the cells, but absorb their food supply by numerous thin-walled, globular haustoria, which are pushed into the cell cavities.

The first new infections always come from swarm spores which must reach the under surfaces of the leaves. A swarm spore which has come to rest produces an infection thread which squeezes through the first stomatal opening it can reach, expands in the substomatal chamber and

then produces more slender branches which penetrate deeper. The period of incubation, that is, the time which elapses from the entrance of the infection thread to the production of evident "oil spots," varies from 5 to 20 days, due to seasonal conditions and differences in the susceptibility of varieties, 7 days being a fairly common incubation period for susceptible varieties.

After a period of mycelial development, the hyphæ become massed in compact cushions just beneath the stomata and, under favorable condi-

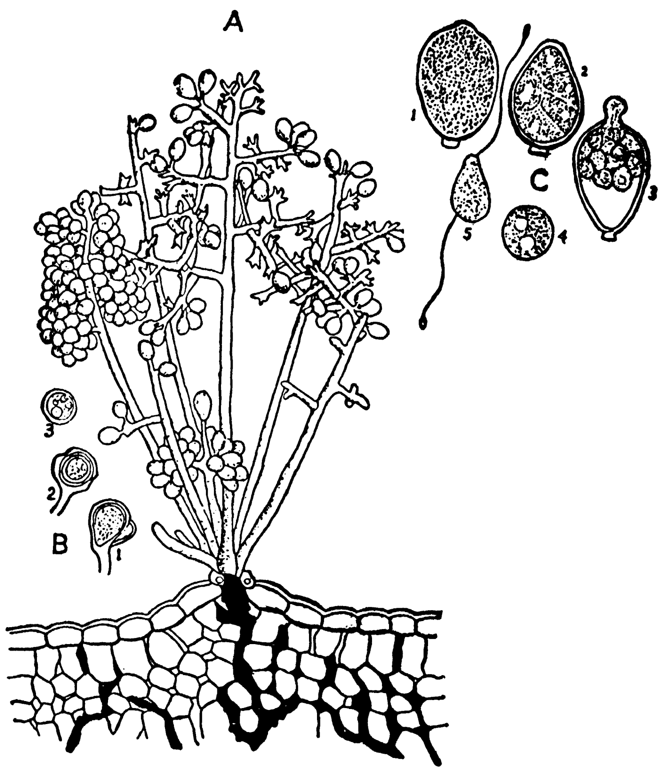

FIG. 125.—Downy mildew of the grape. *A*, conidiophores with conidia emerging through a stoma from an intercellular mycelium (represented as solid black); *B*, three stages in the formation of oöspores; *C*, stages in the germination of a conidium to form swarm spores. (*After Millardet.*)

tions of moisture and temperature, three to six (maximum 20) aerial hyphæ grow out through a stomatal opening and develop into branched conidiophores, bearing numerous conidia (sporangia) or summer spores. Under certain conditions the conidiophores may break directly through the epidermal cells, which are first killed, then crushed and disrupted by the growth of the fungus. Each conidiophore produces three to six main branches, which are, in turn, branched several times, the terminal branches ending in two to four short, slender sterigmata or spore-bearing

tips. Large numbers of these conidiophores massed together give the characteristic downy character to the under surface of the leaf lesions, or cause the "gray mold" of fruits.

Each sterigma can produce but a single spore, which is formed by a swelling at its end, which later becomes separated by a cross-wall. A single nucleus which passes into the developing spore divides later to make the conidium multinucleate. The conidia vary in size, the average being 11 to 18 by 15 to 31μ, with others reaching 40 to 50μ; the majority are ovoid, some are nearly globose, while others are long and narrow. The numerous conidia give to a fully formed conidiophore an appearance not unlike a miniature bunch of grapes. Infected leaves which show few or no conidiophores will produce a copious crop in 12 to 20 hours if placed in a moist chamber.

When conidia are brought into favorable conditions for germination, there is a slight increase in size due to imbibition of water, and each nucleus with its adjacent protoplasm becomes organized into a swarm spore. These swarm spores separate slightly, begin a movement and soon slip out through an opening that is formed at the terminal papilla. The swarm spores then separate from each other and swim away as naked, fully formed, two-ciliate spores. They are planoconvex, and the two cilia originate from the middle of the flat side. After a period of activity they settle down, become more or less rounded, absorb their cilia secrete a delicate cell membrane and soon send out a protuberance which develops into an infection thread or germ tube, which turns abruptly into the first stoma that it reaches. Unless the infection threads reach stomata, they will perish and no infection will result. This would mean that infections must be through the under surfaces of leaves, since the upper side of grape leaves is generally devoid of stomata.

Sometime after the initial infections (not in autumn only) thick-walled resting spores, the oöspores, are produced in the intercellular spaces of affected parts. The detail of development is not entirely clear, but antheridia and oögonia are formed, the egg apparently becoming uninucleate, as in *Albugo candida.* Antheridia have not been observed so frequently as the oögonia, so it is uncertain whether fertilization is necessary. The fully developed oöspore is 25 to 35μ in diameter, filled with a granular protoplasm containing conspicuous oil globules, and surrounded by a thick but somewhat roughened epispore. The oöspores behave as in typical downy mildews and remain dormant until the following spring, when germination takes place. Only oöspores which have been subjected to the freezing winter temperatures are capable of germinating, but some individual spores have been found to require exposure through a second winter to complete their rest period (Arens, 1929).

Under normal field conditions the oöspores will be set free by spring by the disintegration of the tissue of the diseased structures in which they

are formed. Under suitable moisture and temperature conditions the oöspores germinate by the production of a short unbranched promycelium which bears a single large conidium or sporangium. The temperature range is 13 to 33°C. with the optimum 25°C (Arens, 1929). This conidium produces swarm spores in the manner already described. These spores, or the conidia previous to germination, are splashed by rain to the lower leaves of the vine and originate the first lesions, which soon develop conidia, that are wind borne, and thus the disease spreads to new leaves or to other uninfected vines.

In both susceptible and resistant hosts the zoöspores collect around the stomata, and penetration occurs. The developing mycelium is at first confined to the intercellular spaces, but in susceptible species, haustoria are soon formed. In the resistant species the hyphæ make no further growth, and these and some of the surrounding host cells perish, causing a more or less marked discoloration, the so-called "sub-infections" (Arens, 1929; Lepik, 1931).

Climatic Relations.—The occurrence and the spread of downy mildew are greatly influenced by climatic conditions, temperature and humidity of the air being the most important factors. The reports of different workers concerning the effect of temperatures on conidial germination are not entirely in agreement, but this feature has been recently investigated by Gregory (1915). His results may be presented in tabular form:

80 to 90°F	No germination
70°F	40 to 50 per cent germination
50°F	95 per cent germination
35 to 41°F	Very slight germination

Under the cooler and more optimum conditions for germination the swarm spores remain active for a longer period, and conidia held at low temperatures and not offered suitable conditions for germination retain their vitality for 14 to 60 days, while they perish in 4 to 6 days in a warm dry air. Much better conditions, therefore, are offered for infections under low temperatures than when high temperatures prevail.

The retarding effect of dry atmospheric conditions on conidiophore and conidia production has already been noted. If the weather remains hot and dry with plenty of sunshine, lesions established under favorable humid conditions may fail entirely to produce the characteristic aerial conidiophores, and consequently under such conditions there is no possibility for the disease to become epiphytotic. Sporulation also depends on temperature, being very slight below 55 and above 82°F. Regions with prevailing dry winds are likely to be relatively free from mildew, while those localities with heavy dews and high air humidity are very subject to the disease if temperature conditions favorable for spore germination also prevail. The production of conidiophores and conidia

reaches its maximum under humid, cloudy conditions when the vines remain moist for some time, while this presence of moisture on the leaf surfaces is essential for the germination of the conidia and also facilitates the migration of the swarm spores. It has been pointed out that the serious attacks of 1900, 1910 and 1915 in France, which reduced yields so greatly, coincided with heavy rainfall and frequent storms, with temperatures favorable for conidial formation (Cadoret, 1927). Heavy rains, if of short duration and followed by conditions which cause rapid drying, are not favorable to the disease.

Infection is influenced by environmental factors operating upon the host, as well as upon the pathogene. Open stomata and an abundance of soluble food in the leaf tissue favor infection, if temperature and moisture conditions are favorable for germination of the fungous spores. No correlation has been found between the number and size of the stomata and resistance and susceptibility (Lepik, 1931). It has been pointed out (Pantanelli, 1920) that both soil moisture and atmospheric humidity affect the opening of the stomata and thus influence infection. With soil of 15 per cent or less of moisture, 80 per cent or more humidity is required for stomata to open, while with 20 per cent or more of soil moisture stomata will open if the humidity of the air does not go below 40 per cent. High humidity of the air favors infection also, because under such conditions the host tissue will have a higher proportion of soluble carbohydrates, nitrogen and phosphorus.

Young leaves are not infected for two reasons: (1) the stomata are closed; and (2) the tissues contain but little sugar or starch and almost no soluble nitrogenous compounds.

Host Relations and Variety Resistance.—The downy mildew has been found to a greater or less extent on practically all wild and cultivated grapes. There seems to be little, if any, difference in susceptibility of the smooth-leaved species and those with a downy lower surface. *Vitis vinifera*, the cultivated grape of Europe, is more susceptible than the native American varieties. Large numbers of hybrids of *Vitis vinifera* with more resistant American species have been produced in France in recent years in the effort to secure other desirable qualities combined with resistance. The success of these efforts may be somewhat judged by a recent classification of black and white hybrids (Pee-Laby, 1926) as follows: (1) those which do not require spraying for either downy mildew (copper) or powdery mildew (sulphur); (2) those resistant except in epiphytotic years; and (3) those requiring but few treatments and giving heavy yields of good quality. It has since been the experience in France that some hybrids which had not required treatment for 10 years were very severely affected in the epiphytotic of 1927, while some remained immune. In the eastern United States, the four most important commercial varieties, the Concord, Niagara, Catawba and Delaware, are

derivatives of our native wild species. The last, which is thought to be part *vinifera*, is more subject to mildew and other troubles. The disease is not confined to Vitis species, but is known on woodbine or five-leaved ivy (*Parthenocissus quinquefolia*) and Boston ivy (*P. tricuspidata*).

Control.—It is now generally recognized that the oöspores offer the general and probably the only means of carrying the fungus over the winter, since the conidia are short lived and the mycelium does not persist in either buds or fallen leaves. Control practices must then be directed to the cutting down of the first sources of infection (sanitary measures) and preventing the germination of the first spores which reach the susceptible parts of the host (spraying). Whenever conditions are such as to permit plowing or cultivating that will bury the surface débris containing the overwintering oöspores, the practice is to be recommended, but principal reliance should be placed on spraying. Spraying cannot kill the mycelium after infections have taken place, since the fungous body is internal rather than external as in the powdery mildew. The use of fungicides must therefore be preventive rather than curative in effect.

Since mildew varies so greatly in severity in different regions, the spraying program must of necessity vary. In the less favorable localities (or for resistant varieties) no spraying will be necessary, but in other regions two to six sprayings may be necessary. If the mildew is not very severe, three sprayings as follows will give satisfactory protection:

1. When the shoots are 6 to 8 inches long.
2. Just after blossoming.
3. Before the fruit changes color.

When additional sprayings are deemed necessary, they should be timed in agreement with the development of the fungus and the condition of receptivity of the vines, both of which are influenced by weather conditions. If infections in the "oil-mark" stage are present, they will not be a source of danger until humid conditions prevail, so spraying should precede probable rain periods. When vegetative activity slows down, that is, when cane elongation is retarded, the host is more susceptible. Cool weather is the principal retarding factor, and if it corresponds with a falling barometer, or the weather service predicts rain, spraying would be in order. Control in epiphytotic seasons still seems to be rather unsatisfactory in France. Consecutive applications of the fungicide have been recommended for the first and second spray periods in regions of severe infestation.

Copper sprays have given best results in the control of mildew, Bordeaux or Burgundy mixture being used, with Bordeaux as first choice. Many other formulæ as well as proprietary preparations have been tried in recent years. In some tests the following order of effectiveness has been reported: Bordeaux, nosprasen, kurtakol, nosperal, Horst dust and nosperit. The strength of Bordeaux has varied in actual practice,

5-5-50 giving good results with medium severity, but even an 8-8-50 strength has been recommended for violent invasions. Since it has been shown that zoöspores live well in acid media but not in alkaline media, fungicides should not be used which are distinctly acid or which will develop acid under the operation of atmospheric agencies.

Some copper-containing dusts have been used, either alone or mixed with sulphur, if powdery mildew is also present. They do not adhere so well, and have not afforded so perfect protection as the liquid fungicides. They may, however, be used as a supplement to liquid sprays when wet weather makes repeated treatment necessary. Successful spraying should reduce the loss to 1 per cent or less. There are some indications that susceptibility to downy mildew may be reduced by fertilizers, such as phosphoric acid, potash or lime, while rapidly growing plants with less concentrated cell sap are more susceptible.

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IMPORTANT DISEASES DUE TO DOWNY MILDEWS AND ALLIES

SAPROLEGNIACEÆ

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ALBUGINACEÆ

White rust of cabbage, radish and other Cruciferæ (*Albugo candida* (Pers.) Rous.).—(See special treatment, p. 432.)

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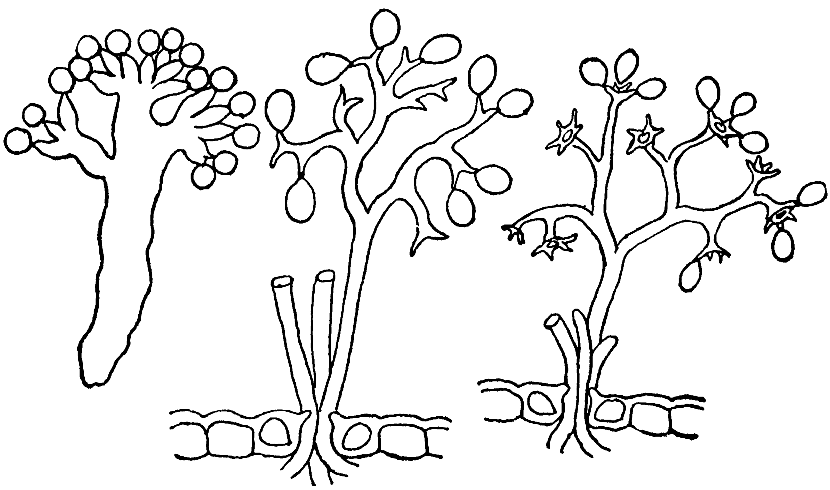

A B C

FIG. 126.—Conidiophores of downy mildews. *A*, Sclerospora; *B*, Peronospora; *C*, Bremia.

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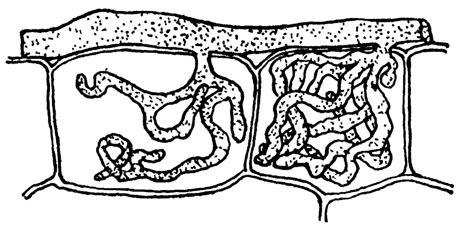

FIG. 127.—Branched haustoria of a Peronospora. Compare with Fig. 121*A*. (*After De Bary.*)

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CHAPTER XVII

DISEASES DUE TO CHYTRIDS

CHYTRIDIALES

The species belonging to this order are mostly obligate parasites of very primitive character which live on other microscopic organisms, such as protozoa, rotifers, algæ, water molds and some other fungi, pollen grains that fall into the water and a smaller number which live in the cells of seed plants. Many of the seed-plant hosts are aquatic forms, but a few are terrestrial plants of economic importance and suffer serious disturbances from the attacks of these minute forms. The name of pond-scum parasites is sometimes applied to the order, because of the large number of species which attack fresh-water algæ.

General Characters.—The fungous body consists of a single cell or a single cell with a few root-like or hypha-like outgrowths. In a few genera a plasmatic body of delicate fibrils penetrates from one cell of the host to another, to form a plasmacomplex, or a branched mycelium may be formed. The plant body generally organizes the reproductive stage or passes into a resting form after a short existence in the vegetative condition. The fungous body may be transformed directly into a zoösporangium, or zoösporangia may be formed from specialized portions of the plant body. The zoösporangia are either thin walled and capable of organizing zoöspores or swarm spores at once, or they are thick-walled resting sporangia, which develop swarm spores only after a period of dormancy. The swarm spores are mostly uniciliate, occasionally biciliate and generally of definite form, although those of a few species have an amœboid character.

In most species asexual reproduction by swarm spores predominates. In some forms two separate cells unite, the contents of one passing into the other, to form a zygote or zoösporangium, or in another case resting sporangia are formed by the conjugation of two swarm spores.

The Chytridiales are probably primitive forms which occupy a place close to the base of the fungous family tree, but the opposing view sees them as degenerates in which the power of sexual reproduction has generally been lost.

The order includes six families and many genera, of which the following are the most important as furnishing parasites of crop plants:

Plasmodiophoraceæ: Plasmodiophora and Spongospora.
Olpidiaceæ: Olpidium and Olpidiaster.
Synchytriaceæ: Synchytrium.
Cladochytriaceæ: Physoderma and Urophlyctis.

Plasmodiophora.—The vegetative body within the host is a naked protoplast, at first uninucleate, but increases in size and becomes multinucleate within the host cells. These form free spherical, smooth-walled uninucleate resting spores, which when set free germinate to form amœboid zoöspores (myxamœbæ) by which new infections result.

Spongospora.—The vegetative body is a multinucleate naked, plasmodium-like body. The resting spores are grouped in sponge-like balls, consisting of many uninucleate cells. Each cell of the spore ball germinates to form a single, uninucleate amœba (myxamœba), and these fuse to form plasmodia-like bodies previous to penetration of a new host.

Olpidium.—The vegetative body is a naked protoplast which later surrounds itself with a thin cell wall and becomes a zoösporangium. It then forms a long tubular neck extending from the host cell to the outside through which the uniciliate swarm spores are set free. Resting sporangia with thicker walls may be formed, but these also germinate to form swarm spores.

Olpidiaster (*Asterocystis*).—Very similar to Olpidium, but the zoösporangium not forming a neck. Resting sporangia showing a star-like folding of the wall, hence the old name Asterocystis.

Synchytrium.—The vegetative body of the fungus is a large cell occupying an epidermal cell of its host which it nearly fills. The content is generally colored yellowish or yellowish red by oil drops. This vegetative cell, which is early surrounded by a cell wall, may be transformed into a resting sporangium or it may divide to form a group of sporangia, a sporangial sorus. The sporangia germinate to form uniciliate swarm spores. The invaded host cell is frequently enlarged and surrounding cells also hypertrophied to form galls, which are generally bright colored.

Physoderma.—Fungous body consisting of fine fibrils and enlarged portions ("Sammelzellen") occupying the host cells and frequently spreading from cell to cell by means of the fibrillar hyphæ. Colored resting zoösporangia formed from the "Sammelzellen," and set free by the disintegration of the host cells. Zoösporangia germinate to form uniciliate swarm spores.

Urophlyctis.—Fungous body of fine branched hyphæ provided with enlargements; confined to a single hypertrophied host cell or spreading to many cells. Resting cells formed by proliferation from segmented turbinate cells and globular or ellipsoid-flattened, and towards one side provided with a circle of hyaline appendages (haustoria) which disappear with maturity. Parasitic on the subterranean or aerial organs of higher plants, and frequently forming conspicuous malformations or galls.

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CLUB ROOT OF CABBAGE AND OTHER CRUCIFERS

Plasmodiophora brassicæ Wor.

The cabbage and other species of the mustard family are frequently attacked by a disease which first produces swellings or distortions of the root, followed frequently by decline in vigor or by death of the affected plants. The characteristic effect upon the root system has suggested such names as "club foot," "clump foot," "club root," "clubbing," "finger-and-toe disease," "anbury," "Kohlhernie" (German), "maladie digitoire" and "gros-pied" (French).

History and Geographic Distribution.—The origin of the disease is uncertain, but it was the cause of concern in Scotland nearly 100 years before it attracted special attention in Russia, where it was so widespread and destructive in the region of St. Petersburg that the Russian Gardeners' Association offered a prize for its study in 1872. As a result Woronin began the study of the disease in 1873 and by 1876 had completed studies which were published in the fall in 1878. Previous work by Caspary had failed to establish the presence of a parasite as the cause of the disease, the true nature of the disease as due to a simple slime mold being first determined by Woronin. Previous to the work of Woronin (1878) finger-and-toe was frequently discussed in garden literature between 1800–1860 in various English journals. As late as 1853–1854, Buckman claimed that the disease was due to a reversion to the original wild forms, while other writers held that soil and climatic conditions were responsible. The disease has become world wide and attention has been given to it in England and various European countries, while it has been frequently reported in American disease literature. Recent important contributions to our knowledge of the disease have been made by Lutman (1913), Cunningham (1914), Chupp (1917), Kunkel (1918), Bremer (1924), Jones (1928), Cook and Schwartz (1930) and Wellman (1930). The disease has given most concern because of its attacks on cabbage and turnips. In 1892, club root was common in New England and the Middle Atlantic states where it was considered one of the worst enemies of the market gardener. It spread southward and westward, reaching Virginia and the Carolinas on the south and Wisconsin, Illinois and Iowa on the west. It appeared in the truck section of the Puget Sound country in sufficient abundance to call forth a special bulletin in 1910. It is now known in 36 states and is reported as important in 21 (Wellman, 1930).

Symptoms and Effects.—The disease may affect seedlings, which after 3 to 5 weeks show "flagging," or the leaves assume a pale-green or yellowish color and the roots will show swellings ten to twelve times the diameter of normal ones. Seedlings that are infected early are usually killed before the season is half over, while later attacks are less serious.

The distorted roots fail to absorb nourishment from the soil and are often unable to transfer the plant food and water collected by the healthy roots to the cabbage head or other storage place. The plant starves, little or nothing being stored either in a head, as in the case of cabbage and such plants, or in a succulent taproot as in the case of turnips and radishes.

This functional failure of the root stunts the plant. The outer and older leaves become yellow and sickly, soon drop and, in the case of cabbage and cauliflower, the head is always small. At first they wilt only in the hottest weather or during midday and recover in the cool of the day, appearing perfectly normal the next morning, but as the disease advances, the outer leaves wither and fall one after the other until in severe attacks the whole plant dies, a result noticeable in July or August, but most severe in September (Cunningham).

FIG. 128.—Club root of cabbage, showing swollen and distorted roots and undeveloped head. (*After Cunningham, Vt. Bul.* **185**.)

The swollen distorted roots begin to decay in the soil towards the latter part of the growing season due to invasion by bacteria and soil-inhabiting fungi, and thus the injury is increased.

The injury to cabbage and similar crops caused by this disease can only be appreciated by those who have seen it destroy whole crops. Cabbage, cauliflower and radish suffer most here, but in Europe, where stock turnips or rutabagas are largely raised, these crops suffer severely. Certain Scottish lands were long ago abandoned for raising turnips because of its prevalence, and many American gardeners no longer raise cabbages for the same reason (Cunningham, 1914).

The type of hypertrophy of the root system varies in the different susceptible hosts. Six general types are recognized by Cunningham as follows:

1. Complete clubbing of main and lateral roots—*Brassica oleracea.*
2. Clubs on main roots, laterals free—*Sisymbrium altissimum.*
3. Clubs on lateral roots, main root free—*S. officinale* and *Erysimum cheiranthoides.*
4. Clubs on main and lateral roots with club-free rootlets above the diseased portions—*Lepidium sativum.*
5. Clubs as tumors of the root—*Raphanus sativus.*
6. Dark, decomposing spots on the root—*R. sativus.*

The hypertrophied roots in group one are very frequently somewhat elongated or fusiform, and thus the malformed root system is of such a nature as to be well characterized by the descriptive name of finger-and-toe disease. In turnips or rutabagas the swellings of the roots are very frequently globular and grouped mainly on the laterals, with the fleshy taproot more nearly normal, but in other cases turnips may be so badly clubbed as to present only a group of branched hypertrophied roots.

Attention should be directed to the occurrence of galls or hypertrophies on the root system of Crucifers and many other species, due to the eelworm or nematode (*Caconema radicicola*). These root knots may easily be mistaken for the effects of club root, as superficial appearances are frequently quite similar, but it is generally quite easy to determine the presence of the nematodes in the cortex of the hypertrophied tissues by microscopic examination (see Root Knot, Chap. XXVIII).

Etiology.—Club root was first shown by Woronin in 1878 to be caused by a species of slime mold to which he gave the name of *Plasmodiophora brassicæ* more recently grouped with the Chytrids. The occasional occurrence of bacteria in the tissue of clubs has led to the theory that the disease was caused by bacteria working in symbiotic relationship with the plasmodia of the slime mold, but cultural tests from clubs have failed to substantiate this theory. The bacteria are to be considered only as secondary invaders, which follow rather than accompany the causal organism (Fedotowa, 1929; Cook and Schwartz, 1930). Although pure cultures of Plasmodiophora cannot be maintained, successful inoculations have repeatedly been made by the use of soil contaminated with the spores, or of soil filtrates containing a suspension of the spores and more recently by bringing together cabbage seedlings grown under aseptic conditions and spores obtained directly from the interior of young clubs. This behavior, with the constant occurrence of plasmodia in the cells of affected roots, is sufficient proof of the active pathogenicity of *P. brassicæ* Wor.

According to recent investigations by Chupp (1917), no bacteria can be obtained by cultural methods from roots showing young swellings, but medium-sized swellings yielded a few colonies, and larger galls, especially those with broken epidermis, yielded numerous colonies. It seems probable that, while the bacteria are not the primary invaders, they follow the slime mold, and by their activities bring about the disintegration or rotting of the diseased roots. In so doing they contribute to the injury and cause the spores which have developed within the roots to be set free into the surrounding soil.

The spores which are set free by the decay and disintegration of the diseased roots may germinate at once or after a period of rest. According to Chupp, 1 to 5 per cent will germinate in muck soil filtrate when taken from fresh roots, more will germinate if they have been exposed to a

freezing temperature for 2 weeks or more, while drying also increases the germination if not too severe. The optimum temperature for germination is 27 to 30°C., but germination and infection will take place at 16 to 21°C. if susceptible seedlings are present. The spores are spherical or slightly hexagonal due to crowding, and somewhat variable in size. The following measurements have been recorded by different workers: 1.6μ (Woronin, 1878); 1.8 to 2.2μ (Molliard, 1909); 1.9 to 4.3μ with average of 3.3μ (Chupp, 1917). In the process of germination the spore swells to be one-third larger, bulges on one side, the cell wall ruptures and the content escapes as a naked, uninucleate mass of protoplasm, provided with a single long cilium and a slowly contracting vacuole. The details of form and behavior of these myxamœbæ or swarm spores as given by different observers are somewhat at variance. According to Woronin, they are long, spindle-shaped structures, of variable form, with the single cilium at the narrow end, and endowed with the power of locomotion by the lashing of the cilium and by the protrusion of pseudopodia in characteristic amœboid fashion. As described by Chupp, they are 1.7 to 3. 5μ in diameter, more or less pyriform, with a thick flagellum from the anterior or pointed end, never amœboid, but with definite form, and move entirely by the action of the flagellum. These two different descriptions are explained by the ability of the zoöspores to change form (Wellman, 1930).

The manner in which these myxamœbæ or swarm spores bring about infection has been one of the disputed points in the etiology of the disease. Various observers have found amœbæ or plasmodia containing two to six nuclei in the young infected tissue, but the exact behavior between the uninucleate, free, swarm-spore stage and the several-nucleate plasmodium within the host cells seems to be somewhat obscure. Chupp claims to have found uninucleate amœbæ within the root hairs of young roots, but Kunkel (1918) seems to be of the opinion that these were not the amœbæ of Plasmodiophora. More recently Cook and Schwartz (1930) report that the swarm spores first penetrate root hairs and form a small plasmodium with up to 30 nuclei, each of which organizes a zoösporangium. These then germinate, forming 4 to 6 zoöspores (gametes) much smaller than typical swarm spores. These gametes then migrate to the epidermal and cortical cells of the root and there fuse in pairs, and the resulting zygotes give rise to plasmodia of typical clubs. No evidence of fusion of zoöspores was found by Wellman (1930). Whether the infecting amœbæ are uninucleate or become several-nucleate before penetration into the host cells is relatively unimportant. There is no evidence of the fusion of myxamœbæ previous to penetration, as in Spongospora, and nuclear division must begin very soon after the amœbæ have entered the host cell if it does not begin earlier.

The idea had generally prevailed that infection could take place only through young tissue of roots. Kunkel (1918) has presented rather con-

clusive evidence that "old tissues are readily penetrated by the parasite and that root hairs are by no means necessary to infection." He was able to secure infections whether inoculations were made near to the root tips or at points far back from the regions producing the root hairs. Further, he was able to produce typical clubs by making inoculations on rather old cabbage stems. When infection takes place through young roots, it is possible that the amœbæ may penetrate through root hairs and other epidermal cells of the primary cortex and pass on into the deeper tissues, but it seems probable that this method of penetration is not common, but rather that infection takes place through portions of roots from which the primary cortex has already been lost.

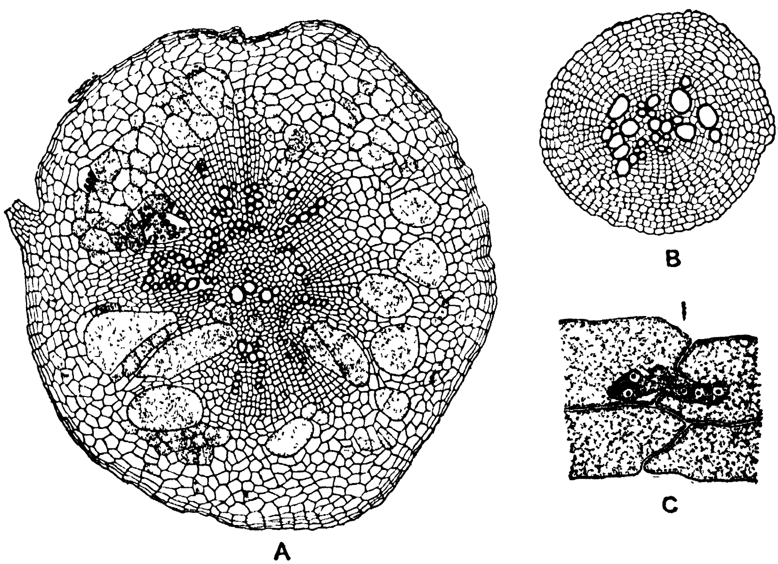

FIG. 129.—*A*, cross-section of a root badly infected with *Plasmodiophora brassicæ*; *B*, cross-section of a young healthy cabbage root; *C*, plasmodium passing through the end of a cell in the region of the cambium. (*A and B after Woronin; C, after Kunkel, Jour. Agr. Res.* **14**, *Plate 70I.*)

Various workers have generally agreed that there are two methods by which the plasmodia may be spread throughout the affected root: first, by the division of cells in which two or more amœbæ are present; and, second, by the migration of plasmodia from cell to cell. First importance has generally been given to the former, with migration as secondary, but recently Kunkel has shown that, in the distribution of the parasite in the tissues, migration is more important than cell division. After the amœbæ are within the cells they begin to increase in size and the nuclei divide, making multinucleate plasmodia which produce new plasmodia by division or separation. In this way a single cell may contain a considerable number of plasmodia. The amœbæ penetrate into deeper-lying cells passing from one cell to another, at the same time migrating some-

what along the longitudinal axes of the root, and finally reach the cambium. They also penetrate into cells on the inner face of the cambium, but migrate more rapidly in the cambium and thus spread the infection lengthwise from the original center of infection. The plasmodia then migrate from the cambium into adjacent tissues of cortex or medullary rays. Carefully prepared sections will show plasmodia passing through the separating cell walls of adjacent cells. As a result of the presence of the parasite certain cells are greatly increased in size, and cell division is stimulated, the stimulus extending beyond the cells actually occupied by the parasite, and in this way a swollen or distorted root is produced.

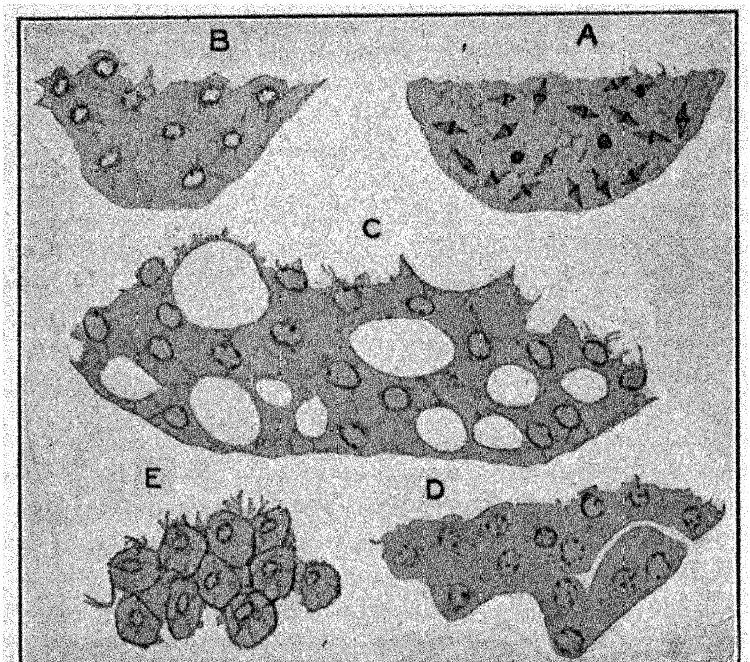

FIG. 130.—Stages in spore formation in the plasmodium. The collection of the cytoplasm around the nuclei is shown in *B* and *E*; large vacuoles are shown in *C*; *D* shows the edge of a plasmodium about ready to form spores; *A*, plasmodium showing nuclear divisions preceding spore formation. (*After Lutman, Vt. Agr. Exp. Sta. Bul.* **175.**)

A single, spindle-shaped club is to be considered as a morphological unit resulting from a single infection that occurred at some point near its middle, rather than as a result of multiple infections.

Finally, some cells remain permanently infected, that is, the plasmodia do not migrate from them. These may be single, in longitudinal chains or in groups called "Krankheitsherde." Affected cells may be crowded full of plasmodia, which are highly granular from the presence of reserve food material in the form of oil globules. Whether these plasmodia finally merge with each other does not seem to be quite clear, but by the time the parasite is ready to start spore formation the individual plas-

modia are packed so closely together as to appear like larger fusion plasmodia. In the formation of spores, the plasmodia become filled with large and small vacuoles, the cytoplasm collects around the nuclei and the whole mass is thus cut up into uninucleate masses which round off and surround themselves with protecting walls. The mature spores are always uninucleate, filled with minute oil drops and contain one or more small vacuoles.

A single diseased root thus produces millions of spores which are set free into the soil by its decay, and these are capable of infecting succeeding crops on the same ground or the disease may be spread to new fields. There seems to be little evidence that the spores are wind borne, although this might seem theoretically possible. The migration of the motile swarm spores in the soil is also believed to be a minor factor in the spread of the disease. It may be spread by soil carried on farm implements, on the feet of animals, by earthworms (Gleisberg, 1922), through the use of contaminated fertilizer, by drainage water flowing from contaminated soils to healthy fields or by the use of infected seedlings.

Host Relations.—The statement is generally made that all species and varieties belonging to the mustard family (Cruciferæ) are susceptible to club root. Although some reports have been published of the occurrence of the disease on other than cruciferous hosts, these appear to have been due to confusing root swellings caused by nematodes (root knot) with the somewhat similar malformations caused by club root. Extensive tests of susceptibility were carried out by Cunningham (1914), using over 100 species belonging to 28 genera. In these tests extending through three seasons, the different species showed a variation from 100 per cent susceptible to 100 per cent immune. The 11 species that remained free from club root are rather rare and very seldom cultivated. Practically all of our cultivated species showed a susceptibility to the disease: varieties of cabbage, cauliflower and brussels sprouts (*Brassica oleracea*), turnip (*B. campestris*), rutabaga (*B. rapa*), rape (*B. napus*), various mustards (*B. spp.*), radish (*Raphanus sativus*), pepper grass (*Lepidium spp.*), alyssum (*Alyssum spp.*) and various other less frequently cultivated species. More recently Naoumoff (1926) has reported the results of infections on 180 species in 49 genera, including the first report on species of the sub-family Thelypodieæ.

Only a few of the more important cultivated species have been tested for comparative resistance of varieties. Of 13 varieties of cabbage tested by Cunningham in 1911, the four showing the most resistance were: Hollander, 26.5 per cent; Stone Mason, 14.4 per cent; Large Late Flat Dutch, 9.9 per cent; and Early Jersey Wakefield, 9.4 per cent free from clubs when grown on heavily contaminated soil. The most susceptible varieties, like Mammoth Red Rock and Perfection Savoy, gave 100 per cent clubbed. Thirteen varieties of radish tested in 1912 and 1913

showed Early Long Scarlet the most susceptible and Early Giant Stuttgart and Early Scarlet Turnip the most resistant. Per cent of clubbing varied from 1.8 per cent in Early Scarlet Turnip to 92.3 per cent in Early Long Scarlet. Turnips and rutabagas were also tested, with the result that Sweet Gorman, White Swede, Early White Milan, Early Snowball and Purple Top Aberdeen can be counted as relatively resistant, while Southern Curled, Early Purple Top Strap-leaved and Improved Purple Top Strap-leaved may be classed as relatively susceptible. Turnips as a class are somewhat more susceptible than the rutabagas, although some varieties of turnips are more resistant than certain varieties of rutabagas (see also Gleisberg, 1923; Davies, 1928).

Predisposing Factors.—Club root is especially favored by acid soils, the number of zoöspores liberated reaching the highest numbers under such conditions. Spore germination and infection are not exclusively dependent upon the H-ion concentration, but no infection will ordinarily result above pH 7.2 to 7.4 (Chupp, 1928). A survey of 116 fields (Wellman, 1930) showing club root gave a range of pH 5 to 7.8. The excessive use of acid fertilizers or highly nitrogenous manures and the withdrawal of lime by the action of smoke gases in industrial centers may be expected to favor the development of club root (Bremer, 1924).

Spores germinate at a temperature range of 6 to 27°C. with the maximum germination at 25°C. Club-root development occurs from 12 to 27°C., with the optimum from 18 to 25°C. "The optimum temperature for host-root development, 20°C., is distinctly lower than the optimum temperature for spore germination and disease development, 25°C." (Wellman, 1930).

The moisture factor seems to be more important than temperature, in its relation to infection. Experimental tests have shown that the disease does not develop where the moisture content of the soil is down to 45 to 50 per cent of its water-holding capacity but will occur when the moisture content is higher, reaching heavier infection as saturation is approached. Low-lying, poorly drained soil might then be expected to favor club-root, and well-drained soils to inhibit it. It has, however, been shown that infection of the host results in 18 hours' exposure to favorable moisture relations; consequently heavy, prolonged rains may offer conditions for infection even in the best-drained soils.

Preventive or Control Measures.—The various practices bearing on prevention or control may be briefly enumerated:

1. *Sanitary practices* designed to prevent the contamination of new areas. Diseased roots if fed to live stock should be thoroughly boiled before feeding, since the spores of the organism will survive passage through the digestive tract of animals, and might be carried to the fields with contaminated manure. When plants are grown in seed beds some method of sterilization should be practiced, especially if club root is

known to be prevalent in the environment. Corrosive sublimate, 1 ounce to 10 gallons of water, applied five times to badly infested seed beds has given excellent protection (Chupp). Later trials by others have been varied in methods of application, but in general fair control has resulted (Clayton, 1926; Preston, 1928; Blunck, 1928). Good results from the use of uspulun have been reported (Bremer, 1923; Clayton, 1926; Preston, 1928) but in general, less satisfactory than with mercuric chloride. Control in seed was obtained for three seasons by watering with a 10 per cent solution of washing soda (Osterwalder, 1929). Consideration should also be given to the fact that soil from contaminated fields may be carried on cultivators and the feet of horses, and such transport guarded against as much as possible.

2. *Crop Rotation.*—Cabbages generally grow best on certain types of bottom land or muck soil, and since this type of land is restricted in area in many localities, there is a tendency to grow cabbages on the same land for a period of years. Under such conditions club root is likely to increase in severity until the entire soil becomes heavily contaminated. Since it has been shown that the club-root organism can live in a soil for 3 or more (6) years, a comparatively long rotation should be adopted in handling contaminated soils. Four or five, and preferably six years should intervene between cabbage crops and no cruciferous crop of any kind should be grown in the interim. Attention should also be given to the elimination of all weeds belonging to the mustard family.

3. *Use of Fertilizers.*—Consideration should be given to kinds of fertilizers not to use as well as to those which may be used with profit. The immediate application of barnyard manure is favorable to the disease, hence this fertilizer should be applied during the season preceding a susceptible crop, but not to a cabbage or turnip crop itself. The same rule would apply if acid phosphate is to be used as a fertilizer. Since most soils in which club root becomes severe are distinctly acid, the *use of lime* has long been practiced with marked success, and should certainly be adopted for contaminated soils on which rotations of sufficient duration are not practical. Lime in various forms has been used as raw-ground limestone, caustic lime, air-slacked lime and hydrated lime, the last giving the best results for field use, 1500 to 2000 pounds per acre for commercial control in badly contaminated soils (Wellman, 1930). Best results will be obtained by treatment several months previous to use of the ground. Cunningham reports an increase in yield of cabbage from 672 pounds per acre where no lime was applied to 23,082 pounds per acre when treated with lime. Calcium cyanamide, which is also of value as a fertilizer, has been recommended as a substitute for lime (Kindshoven, 1928).

4. *Drainage.*—Since an excess of moisture in the soil is favorable to the development of club root, low-lying or water-logged soils, whether

acid or non-acid, should be thoroughly drained and the physical condition improved as much as possible, but drainage alone cannot be counted on to control the disease.

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POWDERY SCAB OF POTATOES

Spongospora subterranea (Wallr.) John.

Powdery scab is a disease that attacks roots, stems, stolons and tubers, producing small hyperplastic galls on the first three structures and small, circular, scab-like lesions or, more rarely, cankers on the tubers. Only parts that are underground are infected. Secondary rots may enter the tubers through the scab lesions.

Various common names have been applied to the disease, such as corky end, corky scab, powdery scab, Spongospora scab and potato canker, but powdery scab has been most generally used, especially in America. The name is descriptive of the character of the lesions and serves to distinguish the trouble from the more widespread and common scab (*Actinomyces scabies*). In Germany it has been called "Kartoffelräude," "Knollenbrand" and "Schorf" or "Grind" at various times; in Sweden it is known as "Skoro"; and in Ecuador as "Cara," which is equivalent to the English word "scab."

History.—The disease was first described in 1841 as occurring in Germany. Berkeley discovered the disease in England in 1846 in connection with his studies of late blight, and Brunchorst studied it in Norway in 1886, but did not then recognize it as identical with the trouble known to be in other countries. While the disease was spread more or less throughout Great Britain and on the Continent, it did not attract much attention until many years later. It is worthy of note that Lagerheim found the powdery scab in 1891 in Ecuador, where it seemed to be well known to the natives. In 1913 the disease was found in the United States on potatoes imported from both the Netherlands and Belgium, which would indicate that the disease was well established in those countries. Early in the spring of 1913 it was reported from a number of

provinces of Canada by Güssow, and from Maine in 1913 and 1914 by Melhus. Since then there have been sporadic occurrences of the disease in a number of the other northern states, from Maine to Washington and Oregon, and in one locality in Florida, but it has not spread in these localities to any great extent. The discovery of the powdery-scab organism in 1915 on potatoes from Peru lends additional support to the belief that the disease is endemic in South America. More recently the disease has appeared in Australia, New Zealand, Tasmania and Kenya Colony.

Following the discovery of the disease in Canada and northern Maine, a Federal quarantine was established to protect the other sections that were still free from the trouble. This quarantine prohibited importations of potatoes except under a strict system of certification. A few years' study sufficed to show, however, that climatic barriers were more effective than the most rigid quarantines in preventing the spread of the disease throughout the United States, and the quarantine was lifted.

Symptoms and Effects.—The disease appears first on very young tubers and is then evident as small, slightly raised pimples or swellings

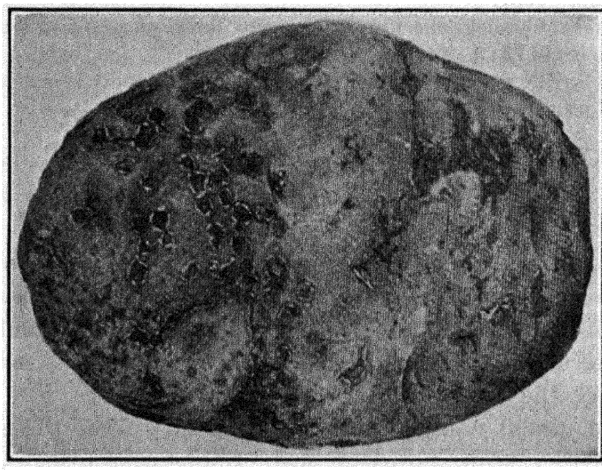

FIG. 131.—Powdery scab of potato showing characteristic appearance of open sori. (*After Morse, Maine Bul.* **227**.)

with a slight discoloration of the surface. The invaded tissue, when cut into, appears purplish. The swellings may occur in groups or patches, or they may be very well scattered over the surface of the tubers. With the progress of the disease, the swellings enlarge, and become somewhat more raised, resembling nodules. Finally, the nodules or swellings break down, leaving a cavity filled with a mass of brownish powder surrounded by the lighter-colored, frayed-out periderm or skin. These scab spots are usually circular or oval in shape, 1/16 to 1/4 inch in diameter, but generally less than the maximum size. They may be few in number or so numerous as to coalesce in groups and thus obscure their normal form. The circular form, the ragged margins of the periderm and the central powdery mass are characteristic features which seem to distinguish the disease from the common scab, in which the lesions are larger, more irregular in shape, sometimes raised, sometimes depressed, but always

lacking the brown powder. The characteristic appearance of the lesions may be modified by friction in handling so that tubers examined in storage may have the top and the powder rubbed away, leaving only slightly depressed, empty lesions. Some lesions may not have matured sufficiently to rupture the epidermis and in many such cases the brown powder may be recognized by breaking away the external covering. It has recently been claimed by Shapovalov (1923) that under certain conditions the lesions of powdery scab become aborted and never advance beyond the simple condition found on young tubers, and that these aborted infections have been described as "skin spot" due to *Oöspora pustulans*. This opinion concerning skin spot does not seem to have found acceptance by English workers.

> In severe attacks, especially in moist soil, a distinctly warty appearance may develop, quite different from the ordinary type of powdery scab. These warts are several times larger than the scab pustules already described, and are usually smooth and roundish, although somewhat irregular, varying in color from a light to a dark brown . . . and more frequently occurring at the terminal or "seed" end of the tuber (Morse, 1914)

It is stated that this "warty condition of the tuber is not so evident after the tuber has been removed from the soil for some time, as the warts become flattened and discolored, so that raised, more or less chocolate-colored scars remain in their stead" (Horne).

In the most severe form of powdery scab, the *cankerous stage*, there is actual destruction of the flesh of the tuber, leaving hollowed-out, eroded areas. These cankers appear to come beneath areas originally occupied by closely aggregated groups of sori of the typical form. The canker stage is generally due to growth in a wet, poorly drained soil, and has been produced experimentally. It is also favored by an excess of lime. The canker stage has not been found in the United States, but it has been reported from Canada and is not uncommon in the British Islands.

The powdery scab affects other underground parts of the potato plant, producing white galls on roots, stolons and stems. These resemble bacterial nodules and vary in size from minute tubercles the size of a pinhead to others as large as garden peas. The extent to which they may be present may be illustrated by a single plant which showed 149 on the roots, 19 on nine stolons and 8 on three stems. The root galls do not appear to exercise any markedly injurious effect upon the growth of the plants, and may be present in certain cases when the tubers are entirely free from infection.

The injury from powdery scab varies with the type of the attack. In mild cases, it consists only of surface scabbing, which disfigures the crop and depreciates its market value, with but little reduction of real value as table stock. The seed value of affected stock is greatly lowered, unless it is to be grown in areas climatically unsuited to the development

of the disease (see Predisposing Factors). In such regions, very severely infected seed has given excellent results. In more severe cases of the common type as well as the canker stage, there may be heavy losses beginning in the field and extending into storage. It is reported that portions of some fields in Maine showed over 90 per cent of the hills affected, while there were several cases in which the infection ranged from 50 to 75 per cent.

The scab sori leave open wounds which permit more rapid loss of water, and serve as the avenues through which wound parasites may enter and cause rot or decay, while in certain cases the scab organism resumes activity and destroys cells adjoining the original lesion. Each of the factors may operate singly or in combination to produce effects which may be designated as powdery-scab dry rot. As a result of desiccation the tissues adjoining sori may become discolored, shriveled

FIG. 132.—Section through a powdery-scab sorus showing disintegrated tissue and numerous spore balls. (*After Melhus et al., Jour. Agr. Res.* **7**, *Plate* 12*A*.)

and shrunken. The renewed activity of the parasite destroys cells in the immediate vicinity of the sorus, producing a hard, dry spot ½ to 1 centimeter in diameter and ¼ to ½ centimeter in depth. This is apparently due to a behavior of the parasite similar to that which occurs in the development of the canker type, but of a milder form. The entrance of wound parasites causes the most destructive type of powdery-scab dry rot. The bottoms of the scab pits are protected by but little or no wound cork, so that penetration of fungi is relatively easy. In Maine, a species of Phoma, described as new (*P. tuberosa*), was the most common of the wound parasites. The lesions caused by this parasite are sunken, dark, often hard and bony, vary from 2 millimeters to 5 centimeters in diameter, may penetrate to a depth of 2 to 4 centimeters and, when removed, leave a clean smooth cavity which has suggested the name of "button rot." Other rot-producing organisms, especially bacteria and

various Fusarium species, may enter the Phoma lesions and complete the destruction with some confusion of symptoms.

Etiology.—Powdery scab is caused by *Spongospora subterranea* (Wallr.) John., one of Plasmodiophoraceæ. While the organism has been recognized as a parasite since the work of Wallroth in 1842, it was some years before its true character was understood. It was first named *Erysibe subterranea,* and even as late as 1850 it was considered one of the smut fungi by Berkeley, as indicated by the name which he gave it, *Tuburcinia scabies.* In 1877, Fischer von Waldheim transferred the organism to Sorosporium, and it was not until the work of Brunchorst in Norway in 1886 that it was placed in the Myxomycetes. More recently the concepts of the relationship of the Plasmodiophoraceæ to

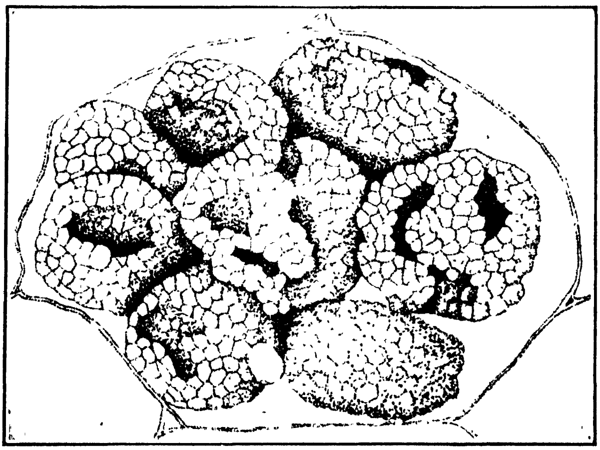

FIG. 133.—Mature spore balls in an enlarged host cell. (*After Osborn.*)

the Myxomycetes have changed, so that the powdery-scab pathogene is now generally grouped with the chytrids.

The powdery content of the scab sori consists of numerous minute brown bodies known as spore balls, which are in reality collections of spores held together in more or less sponge-like, spherical or ovoid masses, varying in size but averaging about 50μ. Under favorable conditions these spores of the ball (all or part of them) may germinate. The germination has been observed by Kunkel on artificial media and it seems probable that the spores will behave in a similar way in the soil or on the surface of young developing tubers. Each cell of the spore ball may produce a single, small, hyaline, uninucleate amœba which generally escapes through an opening in the spore wall, leaving the spore ball intact, but in some cases the entire spore ball disintegrates, setting free as many amœbæ as there were cells in the spore balls. These amœbæ are actively motile and soon crawl away from the spore ball from which they are produced. Under dry conditions, these amœbæ round up and

become surrounded by a thick, rough wall, or become encysted, in which state they are resistant to conditions which would be fatal to the naked amœbæ. With the return of favorable conditions, these cysts or resting cells may germinate and set free the amœbæ again. This behavior will explain how the parasite may persist in the soil from year to year.

The exact manner of infection has been a disputed point, and until the work of Kunkel it was claimed by Osborn and others that single amœbæ entered the host cells and later coalesced to form plasmodia. According to Kunkel, numerous amœbæ external to the host coalesce to form a plasmodium which becomes the infecting body, which then passes down through and between the epidermal cells.

Usually a considerable number of cells are killed at the point where the plasmodium enters. Once beneath the epidermis, it spreads out in all directions and forms a rather flat, disk-shaped mass which separates the epidermis from the tissue beneath. In this way it comes to occupy a space between the uplifted epidermis and sound tissue beneath. Soon, however, a number of projections of pseudopodia begin to extend downward, push in between the cells of the sound tissue and seem to crowd them apart.

According to Wild (1929) the lenticels constitute the principal infection courts, rather than the unbroken skin of the tuber, with some penetration through wounds.

Small strands of protoplasm, the "infecting pseudopodia," are pushed through the softened walls of the host cells, and in some manner become separated from the remainder of the plasmodium. Shortly after the cells become infected they enlarge and elongate radially to five to ten times normal size, forming giant cells which are responsible for the raised condition of young lesions. Ultimately the giant cells are cut up into smaller cells, which are all infected. Finally each nucleus of the intracellular, multinucleate plasmodium organizes a spore, and these become grouped to form the characteristic spore balls. There will then be left in the sorus a group of spore balls, mingled with fragments of old cell walls of host cells or other remains of host cells. It may be noted from this account that the first effect of primary invasion by the plasmodium is the stimulation of the young cells of the tuber to increased cell division, and that later these cells are killed by the plasmodia which become intracellular. In these primary infections, the action of the parasite seems to be limited to a small group of cells.

The plasmodium dry rot which always starts around old sori is due to secondary invasions by plasmodia formed from the germination of spore balls in the base of the sorus. In such cases, the old cells of the tuber with which the plasmodium comes in contact are not stimulated to a new growth, but are quickly killed. The plasmodium enters a cell, consuming its cytoplasm and nucleus and then passes on to the next, leaving behind it a mass of broken-down cell walls, starch grains and other débris. In

this way the dry rot advances by the direct killing effect of the plasmodia, rather than by the production of toxic substances which diffuse into the cells in advance of the line of attack.

The source of an infection is likely to be due to (1) the planting of affected tubers; or (2) to planting clean tubers in a previously contaminated soil. The spore balls carried by infected seed tubers germinate in the soil under the same conditions as the seed, and plasmodia are produced which may cause the infections in the manner outlined above. Spore balls generally remain dormant during the winter, but in the spring many will germinate, and under favorable conditions the plasmodia are probably able to lead a saprophytic life if a susceptible host is not available, or under conditions of stress single amœbæ may become encysted as

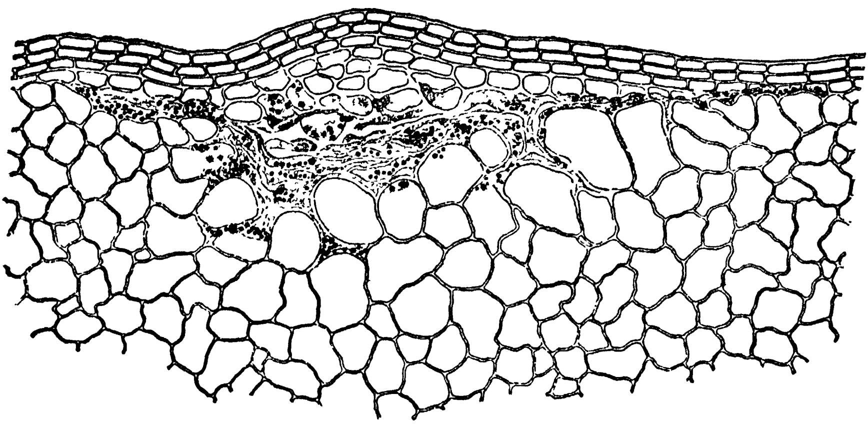

FIG. 134.—A semidiagrammatic drawing of a section through a very young sorus, showing the infecting plasmodium as it pushes down between the cells. (*After Kunkel, Jour. Agr. Res.* **4**, *Plate* 29, *Fig.* 3.)

resting spores and so prolong the life of the parasite. It is uncertain how long the powdery-scab organism is able to live in the soil, but English authorities believe that they have evidence that a new crop may be infected after a lapse of 3 to 5 years.

Predisposing Factors.—Careful study of the powdery-scab disease has shown that infection takes place only under favorable climatic conditions. The essentials for infection seem to be rainfall periods during the young stages of tuber formation, followed by cool, damp, cloudy weather. If in addition the soil is poorly drained, the chances of infection are increased. The proper temperature, coupled with the right moisture conditions, must be provided for infections to take place. It seems that the favorable relations are found only in American sections near the Canadian boundary and farther to the north, with the possible exception of one locality in Florida where winter potatoes are grown. It is worthy of note that heavily infected seed planted at fifteen different places on the Atlantic Coast from Massachusetts to Florida, and at six different

points in Washington from Everett southward, yielded an absolutely clean crop. These and other experiences point to a very effective climatic barrier to the spread of the disease. An ideal soil for infection is said to be one with large pore spaces, a high humus content, a high methylpentosan content and large water-holding capacity. The incidence of the disease is not affected by a pH range of 5.9 to 7.6 (Wild, 1929).

Host Relations.—For many years the potato was supposed to be the only host of *Spongospora subterranea,* but the discovery of galls on the roots, stolons and stems of the potato led Melhus to test the susceptibility of other species of Solanaceæ. Of 16 species planted in contaminated soil, seven developed infections, and it is significant that the tomato was one of the susceptible varieties. In no case were mature spore balls produced in any of the root galls, but this was attributed to the shortness of the season. *Solanum nigrum,* a common weed, remained free from infection.

There seem to be marked differences in the susceptibility of different potato varieties, but it is uncertain whether any are immune. In planting tests in Maine in 1915, four named varieties and seven seedlings remained free from infection, and some varieties showed very slight infections, while others were severely affected. Since the control plantings of the variety, Green Mountain, showed very wide fluctuations in the per cent of infection, it is believed that the variation in varietal response was not due entirely to resistance, but rather to the fact that the tubers of certain varieties escaped infection.

Control Practices.—The following control practices may be emphasized:

1. Select potatoes free from the disease for seed purposes in regions climatically favorable for the disease.

2. Avoid contaminated land. In fields known to be infested, a long rotation (3 to 5 years or more) should be followed as a means of starving out the parasite.

3. If contaminated land must be used, heavy applications of sulphur up to 900 pounds per acre will very materially reduce the disease. Attention to drainage of the land may also be beneficial. Lime should not be used, as it increases the severity of the disease.

4. Infected seed or tubers suspected of being contaminated should be disinfected. No treatment of infected seed has given perfect control, but best results have been obtained by using hot formaldehyde, 2 pints to 30 gallons of water at 46 to 50°C. for 5 minutes, or mercuric chloride, 4 ounces to 15 gallons water at 44 to 45°C. for 5 minutes. These treatments have given better results than the standard long, cold treatments with either formaldehyde or mercuric chloride.

5. Attention should be given to all possible sanitary practices to guard against the introduction or spread of the disease. It should be borne in

mind that the spore balls may be carried by contaminated bags or other articles which have come in contact with infected tubers or with contaminated soil. Farm implements or contaminated manure may habor the organism. Infected tubers or parings may be boiled and fed to hogs.

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PHYSODERMA OR BROWN SPOT OF CORN

Physoderma zeæ-maydis Shaw

This is a disease of corn affecting both culms and leaves, and causing spotting or blight and lodging. Although it has been known only a few years in this country, it has been called by a variety of common names, such as "corn measles," "corn pox," "dropsy," and "spot disease." The terms "rust" and "frenching" have also been incorrectly applied to the trouble. Teosinte (*Euchlæna mexicana*) is the only other host known to be affected.

History.—The first report of the existence of the disease in the United States was made by Barrett in 1912, but since special studies of the trouble have directed attention to it, evidence has been collected of its occurrence at a somewhat earlier date. It is known to have been present in South Carolina in 1911, and the recent survey by the Department of Agriculture renders it probable that the disease was known to farmers long before it attracted the attention of pathologists. It was noted in Mississippi in 1914 and in Florida and Kansas in 1915. The first published description of the disease was by Shaw in 1912, who reported its occurrence in India. The disease has since been found to occur in China, Japan and other oriental countries.

Geographic Distribution.—As a result of a detailed survey by the U. S. Department of Agriculture during the seasons of 1916–1918, the range of the disease is now

well known. The western limit of the disease is marked by a line including the southeastern corner of South Dakota and extending south through Nebraska, Kansas, Oklahoma and Texas. The northern and eastern limits include Iowa, southeastern Minnesota, Illinois, Indiana, Ohio, West Virginia, Maryland, Delaware and New Jersey. The region of greatest prevalence extends from the northern boundaries of Tennessee and North Carolina southward to the Gulf. The disease is less prevalent and severe west of the Mississippi and north of Tennessee and North Carolina.

Symptoms and Effects.—The disease attacks the leaves, both blades and sheaths and also the culms, but is rarely seen on the outer husks of the ear. The first evidence of the disease is the appearance of slightly bleached or yellowish spots, 1 millimeter or slightly more in diameter, which soon become darker, and finally brown to reddish brown, with a lighter margin. Adjacent spots may coalesce, and they may be so numerous as to give the blade a rusty appearance.

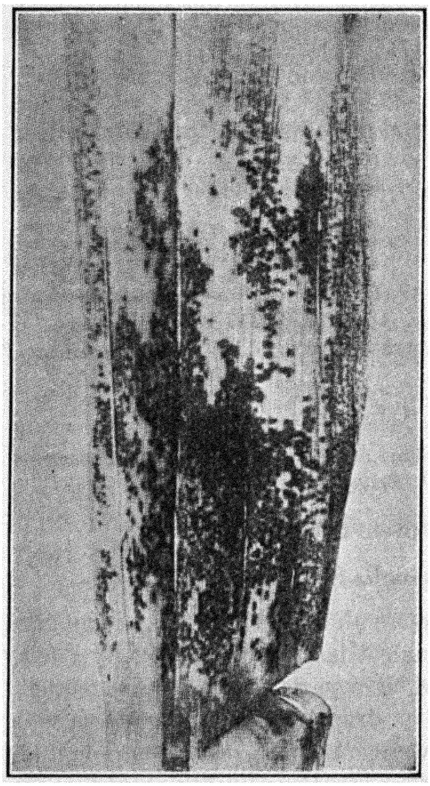

FIG. 135.—Portion of a leaf blade of corn showing the effects of a severe attack by *Physoderma zeæ-maydis*. (*After Tisdale, Jour. Agr. Res.* **16**, 1919.)

The spots on the midrib and leaf sheath are generally larger, up to 5 millimeters in diameter, irregular in shape or almost square, and generally darker than the leaf lesions. The infections may be very numerous, causing the isolated lesions to coalesce and make the entire sheath brown. The brown coloration is due to the death of the host cells and the accumulation of brown spores. The fungous invasions are often accompanied by more or less reddening of the tissues, which may sometimes mask the lesions. The lesions on the culms are most abundant at the nodes and are very similar to those of midribs and sheath.

Towards the maturing period of the host, the epidermis over the lesions dries and becomes loose, where it breaks easily and exposes the brown spore dust, which is readily liberated. The entire parenchyma tissue of the invaded parts may be involved and killed, leaving only the veins or vascular elements, which may appear as separated threads after the spores are liberated. Severe infections on the leaf sheath may kill the leaf before the plant reaches maturity. The lower nodes of a culm may be completely girdled by the fungus, and so weakened that they

break over before they are completely mature. In severe attacks of the disease the spotting and death of leaves and the lodging of the stalks all contribute to the loss, which may be considerable. Based on reduction in yield of grain, the most severe cases have resulted in losses up to 10 per cent, with a material reduction in the forage value of the stalks.

Etiology.—The disease is caused by one of the Chytridiales, which was described by Shaw as *Physoderma zeæ-maydis,* and it has been produced artificially by spraying healthy plants with a suspension of zoösporangia. A new crop of zoösporangia developed after 2 weeks. The brown dust that appears in the older lesions consists of large numbers of separated spore-like bodies which behave as sporangia. A considerable

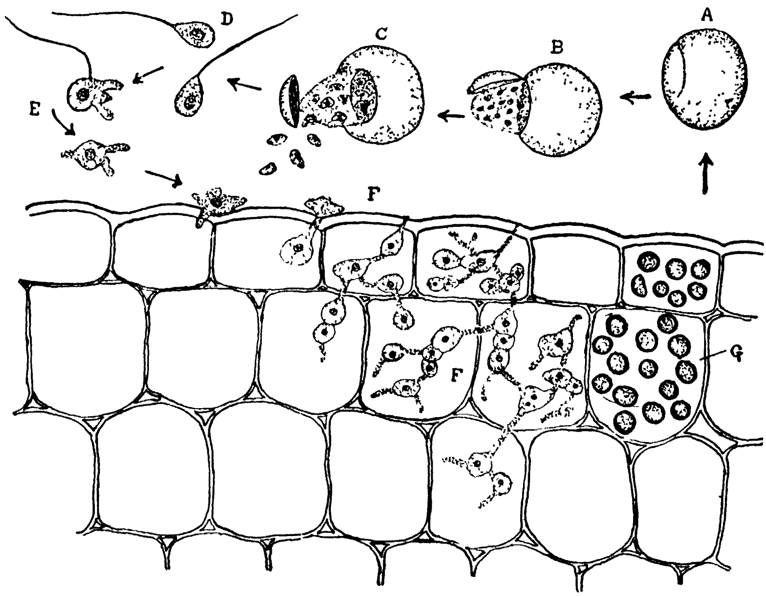

FIG. 136.—A life-cycle diagram of *Physoderma zeæ-maydis.* *A*, sporangium; *B*, *C*, opening sporangia showing early stages of zoöspore formation; *D*, zoöspores or swarm spores; *E*, germinating swarm spores; *F*, successive stages of infection with the development of enlarged cells or "Sammelzellen" and connecting fibers; *G*, host cells filled with mature sporangia. The contents of host cells have been omitted. (*Adapted from Tisdale, Jour. Agr. Res.* **16**, 1919.)

number of these sporangia may occupy each host cell. They are 18 to 24 by 20 to 30μ, provided with a thick, smooth, brown wall, slightly flattened on one side, which is provided with a circular cap or lid. The sporangia pass the winter in the dead remains of infected plants or in the soil and germinate the following season. They may remain as a residual soil contamination or be carried away by such agencies as insects, running water, wind and by various agricultural practices. Under proper conditions of temperature and moisture the sporangium germinates. There is an increase in size by absorption of water, and the lid or cap opens in a door-like fashion, the swarm spores are organized within the

escaping mass (the endosporangium) and finally break through an apical papilla, and swim away. Each zoöspore is 3 to 4 by 5 to 7μ, provided with one long polar cilium, and a comparatively large central oil globule. After a period of activity, a swarm spore settles down, loses its cilium, becomes slightly amœboid and then germinates by the production of fine, fibrous hyphæ.

If the germination takes place on the surface of a susceptible host, one or more hyphæ may penetrate the epidermal wall and then expand within the host cells to form special, enlarged vegetative cells called "Sammelzellen." These groups of enlarged cells (two or more) are always intracellular, and give rise to other slender fibers which give rise at once to other enlarged cells or pass into adjacent cells and there produce other groups of "Sammelzellen." The zoösporangia are formed direct from some of the enlarged cells or at the end of special hyphæ which grow out from them. When sporangial formation is complete, the mycelium has entirely disappeared and the sporangia appear to fill the dead host cells in which they were formed.

Predisposing Factors.—When sporangia are present in a field and suitable moisture and temperature conditions prevail when the corn plants are not more than half grown, the disease is likely to develop in severe form. The temperature factor is the most important, and probably limits the severity of the disease in its northern range and excludes it from other cooler regions. The zoösporangia require a minimum temperature of 23°C. for germination, and the optimum temperature seems to be about 28 to 29°C., a temperature which would be uncommon for night conditions in much of the corn belt. The spread of the disease westward is probably limited by the semiarid conditions which prevail through much of the growing period. High temperatures and abundant and frequent rains through the early growth of the corn crop furnish ideal conditions and explain the range of greatest severity of the disease.

Low wet lands or lands near water are favorable to the disease, while higher well-drained lands are less favorable, especially in seasons of moderate rainfall. At higher mountain elevations in the South the disease may be excluded by the cool summer nights.

Control.—Our knowledge of control measures is very imperfect, but certain practices which have a bearing on the development of the disease may be noted:

1. Since the most severe cases have appeared on land cropped to corn for a number of years in succession, and the sporangia are known to persist in the soil, crop rotation is dictated. The new corn field should be located as far as possible from the old field that produced a diseased crop.

2. The removal of the plants from the field as early and as completely as possible would do much to lessen the supply of infective material for the following season. If an infected crop is fed as stover or fodder, the

barnyard manure should not be used to fertilize land which is to be planted to corn.

No indications of resistance to the disease have been noted, although the selection of disease-free plants offers a possibility of obtaining resistant strains.

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POTATO WART

Synchytrium endobioticum (Schilb.) Perc.

This disease attacks the growing potato and causes the formation of various warty excrescences on the tubers and to a lesser extent on other adjacent parts. On account of its characteristic effect, the disease has received various common names, such as black scab, black wart, warty disease, cauliflower disease, potato canker or cancer and potato wart.

History.—The disease was first briefly described by Schilberszky in 1896 from specimens from upper Hungary, but it is not supposed to occur in that country now, although it has been reported in Silesia, Poland and Czecho-Slovakia. It was apparently known to English growers at least 12 years previous. It was first definitely reported from Great Britain in 1902, in Germany in 1908, in Newfoundland in 1909, in Canada in 1912, in Norway in 1914, in the United States in 1918 and in South Africa in 1926. In the United States the disease has recently been studied by Orton and Kern in Pennsylvania, by Kunkel and by Weiss of the U. S. Department of Agriculture, and the distribution has been determined by a systematic survey carried out by the Federal Plant Disease Survey. During the last few years most of the contributions to our knowledge of the disease have been made by English and German workers.

Geographic Distribution.—Wart became widespread in Newfoundland, but during recent years has declined due to the use of immune varieties. Due to prompt extermination, the disease has not spread in Canada since its first appearance. Since the first report of the disease from Luzerne County, Pennsylvania, in 1918, it has been found in several hundred gardens in three counties of eastern Pennsylvania and in six counties in the western part of the state; also in two counties of West Virginia in 1919, and in the northwest corner of Maryland. The infested districts in Pennsylvania and West Virginia are mining regions, where home gardens constitute the principal cultivated land. It is fortunate that it has not yet invaded any of the important commercial potato districts. It seems probable that the disease was introduced from Europe with heavy importations of potatoes just previous to the enforcement of the Federal quarantine in 1912. The disease has now been reported from the greater part of northwestern Europe, but has perhaps reached its greatest severity in portions of England and Scotland, where it has become a decided factor in potato production. In Germany it is considered of little economic importance, although it is widespread in certain industrial districts and certain writers have been fearful that it will spread into agricultural areas.

Symptoms and Effects.—The disease attacks the tubers principally and produces abnormal warty excrescences, which originate at the eyes. These may vary from slight outgrowths barely visible to the naked eye to larger ones which equal the tuber in size or completely cover it and obscure its normal character. The outgrowths or warts are at first whitish or of the color of young tubers, but later become a rusty brown or almost black; hence the name "black wart." The size and the character of the warts will vary with the severity of the infection:

1. Slight infections may occur as small, simple or compound nodules from the size of a pinhead to that of a pea.

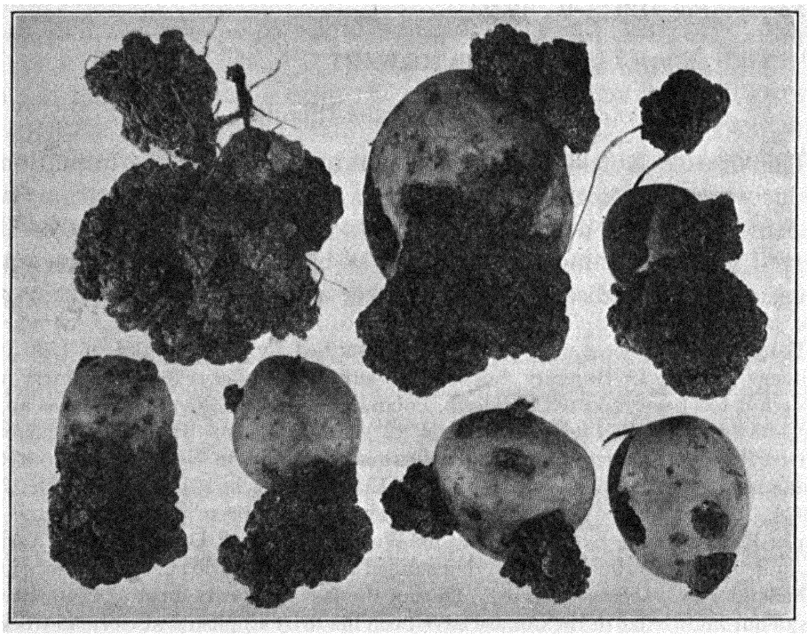

FIG. 137.—Various degrees of wart development on potato tubers. (*After McCubbin Pa. Dept. Agr. Bul.* **394**, 1924.)

2. More severe infections will show one or more fairly large nodular excrescences, each one resulting from an abnormal coral-like growth of the sprouts; hence one of the common names, the "cauliflower disease."

3. In severe or advanced stages the tubers may be completely covered by the abnormal growth and show little or no resemblance to normal potatoes. In this stage the tuber is replaced by this irregular, ragged excrescence.

4. The final and most advanced stage is shown when the affected tuber is reduced to a brownish-black mass which undergoes a soft rot and gives off an odor of decay or dries up.

The warts may occur on all underground parts, tubers, stolons, stems and roots, being most in evidence on the tubers and least frequent on the

roots. Lateral shoots of the stem above ground are sometimes attacked and transformed into dense bunches of minute, leafy or cock's-comb-like outgrowths, while excrescences rarely appear on leaves in contact with infected soil. The disease neither kills the host plant nor seriously affects the growth of the vines, and is generally not evident until digging time, although some observers report that seriously diseased plants remain green longer than normal ones.

The disease may be only slightly in evidence or so serious as to cause a complete loss of the crop. When young tubers are severely attacked, their growth is checked and the whole potato becomes involved. The disease not only reduces the quantity of the crop but the quality as well, since badly warted tubers are unsalable, and subject to decay either in the ground or after they go into storage.

Etiology.—Potato wart is caused by *Synchytrium endobioticum* (Schilb.) Perc., one of the Chytridiales. The organism was first named by Schilberszky in 1897 from specimens from upper Hungary and referred to Chrysophlyctis. In 1910, Percival, an English botanist, studied the disease and referred the causal organism to another genus, Synchytrium of the same order, while Massee called it *Synchytrium solani*, thinking the organism in England distinct from the organism described by Schilberszky. Various other workers have referred the wart organism to Chrysophlyctis but the opinion of Percival has recently been confirmed by the detailed studies of Miss Curtis (1921). Cooke and others assigned the organism incorrectly to *Œdomyces leproides* Trab., which is another parasite belonging to the same order.

The causal organism of potato wart does not form a mycelium but remains in a simple form, consisting at first of a uninucleate prosorus in which nuclear divisions ensue to produce, first, mother cells of sporangia, then within these zoöspore initials. The parasite is confined very largely to the five or six outer layers of cells of the warts, and is set free into surrounding soil by the disintegration of the tissue of the warts. In the matured condition of the warts the parasite is present in the form of sporangia (spores), which are of two kinds: thin-walled summer sporangia, which can germinate at once; and thick-walled resting sporangia, which only germinate after a period of dormancy. The resting sporangia are globular to oval, rusty brown or dark brown, 50 to 70μ in diameter, with thick wall, roughened by irregular ridges. Under favorable conditions the contents of a sporangium organize numerous uninucleate swarm spores which escape to migrate through the soil moisture. These swarm spores are more or less pear-shaped, uniciliate, 1.5 to 2.4μ in diameter, with an actively amœboid body, while the cilium has a rotating movement and also acts as a rudder. They behave either as swarm spores or as facultative gametes. In a suspension of swarm spores, certain ones appear to come to rest while others are still active, the active or male

gametes seeking the passive or female gametes and pairing with them (Köhler, 1930). If they penetrate a suitable host directly, they reproduce the summer sporangial stage and cause only a very limited epidermal hypertrophy. If, however, the swarm spores fuse in pairs before penetration, the zygote develops into a resting sporangium, the effect on the host being to stimulate cell division. In the absence of a host both swarm spores and zygotes soon perish.

The swarm spores after entry into the host may be found as intracellular amœboid bodies, embedded in the living cytoplasm and generally grouped around the nucleus. This plasmodium increases in size until it nearly fills the cell and becomes surrounded by a thin, but distinct, wall. The more mature plasmodia show a reticulate cytoplasm, with numerous

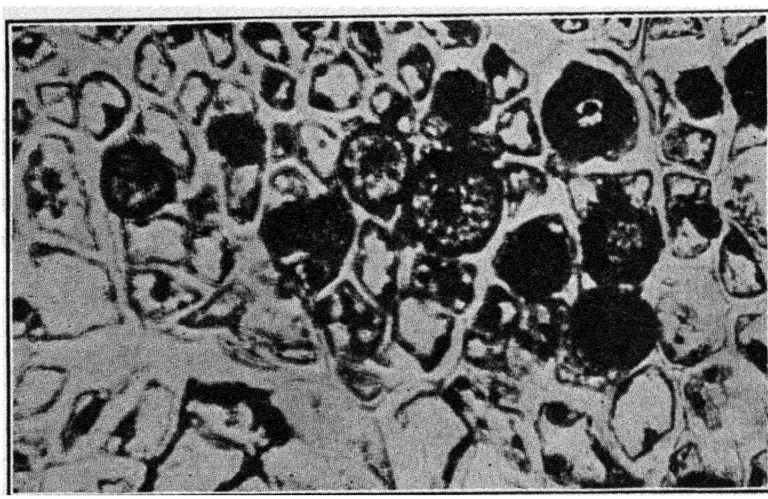

FIG. 138.—Section through an older part of a wart, showing structure and position of resting spores or sporangia. (*After Artschwager, Jour. Agr. Res.* **23**, 1923.)

minute fungous nuclei, while the nucleus of the host cell atrophies at one side of the cell but remains outside the sporangium. The details of the process of sporangium formation are not clear, but it is known that the wall of the sporangium gradually increases in thickness and assumes the characteristic color and markings of the mature structure, thus completing the life cycle.

The resting sporangia are produced in enormous numbers, each infected crop liberating millions into the soil by the decay of the diseased tubers. Unfortunately, these sporangia may retain their power of forming spores for a period of years—according to authoritative reports for 6 to 8 years. With continued production of potatoes on land, each succeeding crop may become more heavily infected until it is no longer possible to produce any sound tubers. Resistant varieties seldom form typical proliferations. In many such cases the infection is so retarded

that the sorus does not reach the migration stage or sporangial formation, thus forming the so-called "sub-infections." It has even been noted that zoöspores penetrate the epidermis of young shoots of Great Scot, an immune variety, and develop for 2 days but then shrink and disappear. There is no anatomical basis for immunity, hence the behavior cited indicates that resistance is physiological.

The rapid spread of wart is retarded by the fact that it does not seem to be carried by the wind, which is such an important agent of dissemination in many other diseases. There are, however, many ways in which the disease-producing organism may be carried into new fields or spread from one locality to another.

The sporangia in the soil or on the tubers must be carried to other soils before new infections can be started. The sporangia may be carried into clean soil by drainage from infested soil, by farm implements used to cultivate infested soil, on the feet of men or animals, by planting diseased tubers or tubers that have been in contact with diseased tubers or by planting sound tubers that have grown in infested soil, by the use of manure from animals to which diseased tubers have been fed and by garbage into which warts or peelings from diseased tubers have been thrown. Any agency that distributes infested soil or infected tubers is sure to spread the disease (Kunkel).

Conditions Favoring Infection.—The infection of the potato by wart is influenced by environmental factors, the most important being soil moisture, soil temperature and soil reaction. These factors have been summarized by Weiss (1925):

Germination of both resting and soral sporangia occurs in water, and there is an indispensable minimum of water for the distribution of the motile cells. If the soil moisture content does not at any time reach saturation, germination is prevented, but if it is constantly near saturation infection is repressed, probably through the reaction on the host. The most favorable condition is periodic flooding, followed by drainage and aeration. Infection may occur, if the temperature is favorable, in soil that is wet at insufficient intervals to afford a normal crop.

The complete thermal range for germination of resting sporangia was not determined, but infection resulted when they germinated between 10 and 28°C. Infection from germinating soral sporangia occurred between nearly 0 and 30°C. When the soil temperature was constantly maintained, infection was limited to the range 12 to 24°C., but with variable soil temperature, as in the field, infection occurs when the mean is about 21°, though the upper range may be as high as 30°C.

The most favorable soil reaction is from neutral to slightly acid, the range being from about pH 3.9 to pH 8.5. The potato tolerates somewhat greater alkalinity but with reduction of yield and injury from other diseases. Germination of sporangia is accelerated by a plentiful supply of oxygen and is better in soil extract than in tap water (Esmarch, 1926). Contrary to some reports,

freezing of resting spores does not lead to their germination so soon as favorable conditions are restored.

Host Relations.—While the potato is the principal host of the wart fungus, it is known to infect several other species of the Solanaceæ. It has been reported on black nightshade (*Solanum nigrum* L.) and on bittersweet (*S. dulcamara* L.) by Cotton, and more recently (1918) on tomato by Kunkel. *Solanum alatum* and *Hyoscyamus niger* were later shown to be susceptible (Esmarch, 1925), while *Solanum nodiflorum*, *S. villosum* and *Nicandra physaloides* have been successfully inoculated (Martin, 1929). Of 50 varieties of tomatoes planted on infected soil, seven became infected as follows: Landreth's Red Rock, Maule's New Imperial, Success, Magnus, Carter's Sunrise, Early Detroit and Burbank. According to Kunkel, the warts were confined to the roots and underground portions of the stems, those on the roots being the size of a garden pea or somewhat smaller, while those of the stem were larger than those on the roots. As a result of later studies Weiss (1925–1928) reports all commercial types of tomatoes susceptible but infections were on stem buds and young shoots, never on roots. It is not believed that the disease will cause serious danger to the tomato, even in the most susceptible varieties, but it is of importance to know that such hosts, if planted in infested fields, might serve to keep the wart fungus alive from year to year. It seems probable that other solanaceous hosts will be found, so that efforts should be made to exclude weeds or cultivated varieties of the nightshade family from infested fields in which rotations are being practiced for the elimination of wart.

Previous to the introduction of the wart disease into America it had been found in European countries that certain potato varieties were either very resistant or entirely immune to the disease. During the season of 1919, Kunkel and Orton tested 29 immune English varieties, a large number of American commercial varieties and a number of promising seedlings developed by Stuart. This work was continued until 1923, when practically all American varieties had been tested and enough seedlings to indicate the manner in which resistance to wart is inherited. The English varieties remained immune under Pennsylvania conditions, while the American varieties were grouped as follows: (1) so badly warted that the crop was practically ruined; (2) varieties moderately warted but not so severely as to ruin the crop; (3) those so slightly warted that the disease could hardly be said to do any appreciable damage; and (4) completely immune varieties. Those belonging to the last class were

> . . . Irish Cobbler, Sutton's Flourball and Early Petoskey of the Cobbler group; Ehnola and Extra Early Sunlight, of the Early Michigan group; Spaulding No. 4 of the Rose Group; Green Mountain and Green Mountain Jr., of the Green Mountain group; Round Pinkeye, of the Peach blow group; and the Keeper variety which has not yet been placed in any group.

The data given by the above workers show that

The susceptibility of American potato varieties varies within wide limits. It is fortunate that a considerable number of varieties are immune, and it is especially fortunate that among this number are to be found some of our most important commercial varieties. But it is a regrettable fact that such valuable sorts as the Rural New Yorker, Early Rose and American Giant should be very susceptible to the disease (Kunkel and Orton, 1920)

It is significant that seven of the promising seedlings proved to be immune, so that we may look forward to the development of other immune commercial varieties. Since the report by Kunkel and Orton the varieties McCormick and Burbank have been proved to be immune. A very complete list of susceptible and immune varieties of the different countries has been published (Foëx, 1925).

The relation of wart to potato varieties offers one of the most striking illustrations of complete immunity of some varieties, and varying susceptibility of others, that is known among plant diseases. Immunity to wart is as constant a character in immune varieties as any morphological feature and is transmitted to the offspring in a definite manner (Salaman and Lesley, 1923).

Eradication and Control Measures.—The first measure directed against this disease in the United States was the *quarantine* of 1912 of the U. S. Department of Agriculture, which excluded importations from countries in which wart was known to exist. This embargo was prompted by the knowledge that the wart disease had already crossed the Atlantic, as it was reported from Newfoundland in 1909 by Güssow. Since the discovery of the disease in the United States in 1918, domestic and local state quarantines have been put in force to check the spread of the disease to new localities and an extensive survey of the United States was conducted by the Federal Plant Disease Survey to find out how widely the disease had been introduced. It is undoubtedly true that the disease was introduced directly with European importations, previous to the quarantine of 1912. The knowledge of the present range of the disease, with the continuation of the quarantines, should serve to confine the disease to the few localities in which it has appeared.

The case of Sweden has been frequently cited as an instance of the extermination of wart in a community by prompt action immediately upon its discovery. Wart was reported in Sweden in 1914, but the infested area was placed under quarantine, the soil chemically treated and potato culture abandoned. There has been no recurrence of the disease. In Canada, likewise, prompt repressive measures put into effect following the introduction of some warted potatoes which were used as seed in 1912 seem to have been effective in elimination of the disease. though in this case the prevalent culture of immune potatoes throughout the area exposed to infection has doubtless been a factor (Weiss, 1924).

Promising results have recently been obtained by steam sterilization, using the inverted-pan method. It has been found that this method was effective in killing the fungus when a pressure of 90 pounds was employed for 85 minutes. It would seem that this method or a modification of it might prove effective for small infested tracts or gardens, but it is too expensive for general use. Resting sporangia are killed if moist in 2.5 minutes at 100°C. or in 2 hours at 60°C. Resting sporangia on tubers are not killed by the standard formaldehyde or mercuric chloride treatments (Weiss, 1928).

A number of treatments making use of mercury bichloride, applied either alone or together with common salt to secure a greater penetration, have been successful in eliminating wart for 3 years. The rate of application varied from ½ gallon of a 1–100 solution per square foot to 4 parts of bichloride, 25 of salt and 50 of water, applied at the rate of 1 gallon per foot. Other chemicals which have been entirely successful in freeing the treated ground from wart for 3 years are Bordeaux mixture 8–8–50, ½ gallon per foot; kerosene, 1 pint per foot; lime sulphur, 1–12, 1 gallon per foot; sodium carbonate, 2 pounds per foot; and sulphur, 2 to 6 ounces per foot (Weiss, 1924).

More recent tests of soil sterilization have been reported (Roach *et al.*, 1925, 1926, 1928, 1930; Hunt *et al.*, 1925; Lemmerzahl, 1930), but the practical difficulties of any method of soil sterilization are so great that the success of this method of control is extremely doubtful.

In localities in which the disease is established, control or preventive measures should be practiced. In the first place it should be noted that disinfection of seed is ineffective, and that even the most careful sorting of infected stock would not eliminate tubers showing slight infections or bearing adherent spores. The following control features may be emphasized: (1) Do not use any seed stock from infested fields; (2) give especial attention to all sanitary measures which will prevent transporting the spores of the fungus from infected to clean fields (see Conditions Favoring Injection); (3) infested land should be cleaned as thoroughly as possible from all potato refuse, and should not be cropped to susceptible varieties of potatoes for at least 8 years, and all solanaceous weeds should be kept out; (4) in case of desire to continue the growing of potatoes on infested land select the best suited immune varieties for planting, or in case of a small area use one of the methods of soil sterilization.

It can be stated as an established fact that immunity to wart is a constant character for several of our best potato varieties, and that for practical purposes in infested districts the culture of these varieties may be taken up and the presence of the disease ignored (Weiss, 1924).

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IMPORTANT DISEASES DUE TO CHYTRIDS

Club root of cabbage and other crucifers (*Plasmodiophora brassicæ* Wor.).—(See special treatment, p. 457.)

Powdery scab of potato (*Spongospora subterranea* (Wallr.) John.).—(See special treatment, p. 467.)

Seedling disease (*Olpidium brassicæ* Wor.).—This causes a damping-off of cabbage seedlings by attacking the stem at or near the surface of the soil. WORONIN, M.: *Jahrb. f. Wiss. Bot.* **11**: 556. 1878. BENSANDE, M.: A species of Olpidium parasitic in the roots of tomato, tobacco and cabbage. *Phytopath.* **13**: 451–454. 1923.

Blight (*Olpidiaster radicis* (de Wild.) Pascher).—This disease attacks the roots of flax and numerous other hosts, causing a blight, "Flachsbrand" of the Germans or "brûlure du lin" of the French. GUYOT, A. L.: Contribution à l'étude systématique et biologique de l'*Asterocystis radicis*. *Ann. Epiph.* **13**: 79–93. 1927. BARTLETT, A. W.: *Olpidium radicicolum* de Wild. and the 'hybridization nodules' of Swedes. *Trans. Brit. Myc. Soc.* **13**: 221–238. 1928. VANTERPOOL, T. C.: *Asterocystis radicis* in the roots of cereals in Saskatchewan. *Phytopath.* **20**: 677–680. 1930.

Potato wart (*Synchytrium endobioticum* (Schilb.) Perc.).—(See special treatment, p. 479.)

Leaf galls (*Synchytrium globosum* Schr. and *S. aureum* Schr.).—These species form small leaf galls, the former on violet, wild strawberry, blackberry and a number of other hosts, the latter on such widely separated genera as Viola, Trifolium, Ulmus, and many others, about 130 species being recorded as hosts.

Cranberry gall (*Synchytrium vaccinii* Thomas).—Small, reddish galls are formed on the stems, leaves, flowers and fruits of the cranberry and other related species. SHEAR, C. L.: *U. S. Dept. Agr., Bur. Plant Ind. Bul.* **110**: 37–38. 1907. Also *U. S. Dept. Agr. Bul.* **258**: 12, 41. 1931.

Physoderma or brown-spot disease of corn (*Physoderma zeæ-maydis* Shaw).—(See special treatment, p. 475.)

Crown wart of alfalfa (*Urophlyctis alfalfæ* (Lagerh.) Mag.).—This disease is characterized by the formation of galls, varying from the size of a pea to others several inches in diameter, located at the base of the stem or on adjacent roots. JONES, F. R. AND DRECHSLER, CHARLES: *Jour. Agr. Res.* **20**: 295–323. 1920.

Beet-root tumor (*Urophlyctis leproides* (Trabut) Mag.).—In this disease galls of varying sizes are formed on the roots, very similar in character to those in alfalfa crown wart. MAGNUS, P.: On some species of the genus Urophlyctis. *Ann. Bot.* **11**: 87–96. 1897. Ueber die in den knolligen Wurzelauswüchsen der Luzerne lebende Urophlyctis. *Ber. Deutsch. Bot. Gesells.* **20**: 291–296. 1902.

CHAPTER XVIII

DISEASES DUE TO THE BLACK MOLDS AND ALLIES

ZYGOMYCETES

The members of this group are forms with a much-branched, multinucleate, non-septate mycelium and reproductive structures in keeping with the terrestrial habit which they have adopted. The hyphæ are frequently inflated or constricted rather than uniform in diameter. While the absence of cross-walls is the general rule, they are sometimes formed to separate young portions of the mycelium rich in protoplasm from old parts devoid of living contents. Cross-walls must also be introduced when reproductive cells are formed. Under abnormal conditions the mycelium of some species may break up into isolated cells which behave much like yeast cells.

Asexual Spore Formation.—This is by one or the other of two methods: (1) non-motile spores in aerial spore cases or *sporangia;* or (2) non-motile spores, conidia, borne free on aerial conidiophores. A few species produce both sporangiospores and conidia. The sporangiophores are simple or variously branched, and the sporangia produce from a few to many spores, the number being indefinite. The conidiophores are also simple or branched and the conidia borne singly, or in chains. Some species are also able to form *chlamydospores* (see p. 400).

Zygospore Formation.—Sexual reproduction is by the union of two equal and similar gametes or sex cells to form a *zygospore.* In the formation of a zygospore two hyphæ come in contact with each other. A papilla-like protrusion is formed from each hypha at the point of contact, and these continue to grow in length to form the *progametangia.* Each progametangium cuts off an end cell, a *gametangium,* containing a *cœnocytic gamete.* The cell walls of the two gametangia are dissolved at their point of contact, the two gametes fuse and the product of the fusion surrounds itself with a thick brown wall to form the *zygospore,* supported by the enlarged remaining portions of the progametangia, now called the *suspensors.* A zygospore germinates by the formation of a hypha, which soon gives rise to one or more primary sporangia. A gamete that fails to unite with another one may sometimes become transformed into a spore very similar to a true zygospore and then is called an *azygospore.*

There is no morphological differentiation between male and female mycelia or hyphæ, but it is known that physiological differences exist. The uniting gametes may be formed on different branches of the same

mycelium or they may be produced by separate mycelia. The former are the so-called *homothallic* types, the later the heterothallic types. The sex cells or gametes may be designated as plus (+) or minus (−), fusion occurring only when opposites come in contact. Certain species produce only homothallic mycelia, while others are always heterothallic, and in the latter case two separate strains (+ and −) must be brought together upon the same substratum before zygospore formation can take place.

Two orders of Zygomycetes are recognized:

Entomophthorales.—The asexual spores in this order are conidia only. Most of the species are parasitic upon insects, and cause epizoötics of adult flies or of the larval stages of various moths (*e.g.*, *Empusa muscæ* Cohn of the common house fly; *Entomophthora sphærosperma* Fresen. on larvæ). One species, *Completoria complens* Lohde, is parasitic on the prothallia of ferns.[1]

Mucorales.—Either conidia or sporangiospores are formed by species of this order, but sporangiospores are the more common. Most of the species are saprophytes or weak parasites, but a few are obligate parasites. Most of these are unimportant, as they are parasitic on the mycelia of other fungi. In the seven families only two genera, **Rhizopus** and **Choanephora,** are of importance as furnishing parasites of crop plants, the former producing only sporangiospores, the latter, both sporangiospores and conidia.

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RHIZOPUS DISEASES

Rhizopus nigricans Ehr.

The same organism is responsible for the rotting of various fruits, produces a soft or watery rot of tomatoes, cherries and strawberries,

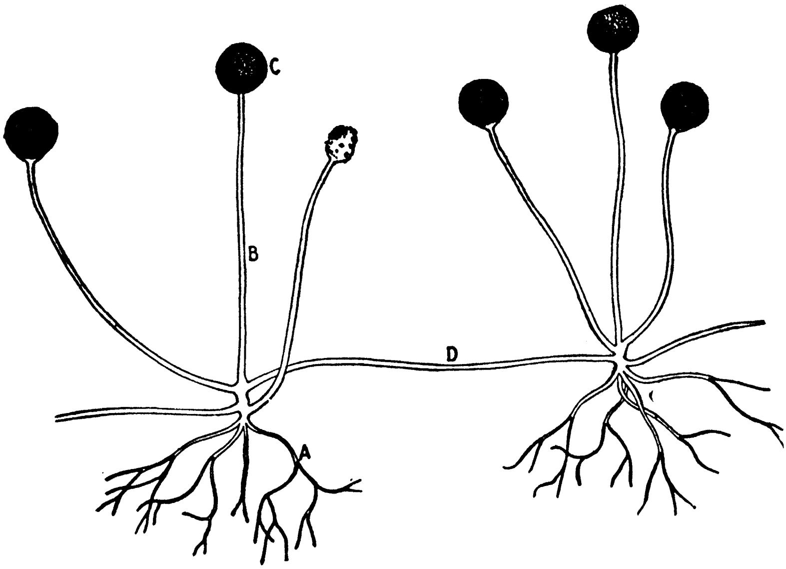

FIG. 139.—General habit of Rhizopus. *A*, root hyphæ, which penetrate the substratum; *B*, aerial hyphæ or sporangiophores bearing terminal sporangia *C*; *D*, a stolon. (*After Sinnott.*)

known as leak, plays a part in causing leak of the Irish potato, causes a soft rot and ring rot of sweet potatoes and attacks seed and seedlings on the germinator.

The Organism.—Two types of spores are produced: asexual spores in sporangia; and sexual spores or zygospores, according to the general type, for the Mucoraceæ. The development may be traced beginning with the asexual spores. Under suitable conditions of moisture and temperature these spores germinate, a hypha generally growing out from opposite poles. These hyphæ show a granular, richly vacuolated protoplasm, remain without cross-walls and branch and rebranch until an interlacing tangle of mycelium is developed in the substratum. Up to this point the growth has been purely vegetative and in the substratum,

but under suitable conditions groups of erect, aerial hyphæ will grow up from the substratum. Some of these hyphæ or *stolons* soon bend over, enlarge slightly at the end and, if they come into contact with the substratum, give rise to a cluster of branched hyphæ which penetrate the substratum, while other branches from the swelling grow up erect, and each develops a spherical globular enlargement, the *sporangium*, white at first but black as it becomes mature. From the base of each cluster of erect, aerial sporangiophores, one or more stolons may arise which grow out, "strike root" and develop another group of sporangiophores. This process may be repeated until the fungus has spread over the surface of the substratum. In this typical condition the fungus becomes differentiated into the vegetative or root hyphæ, distributive hyphæ, or stolons, and the aerial sporangiophores which provide for spore formation.

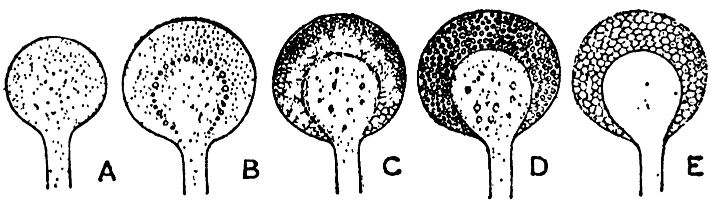

FIG. 140.—Diagrams showing the method of the columella and spore formation in *Rhizopus nigricans*. *A*, a young sporangium; *B*, showing the dome-shaped layer of vacuoles outlining the columella; *C*, showing early stage of cleavage; *D*, showing the contraction stage; *E*, expansion or polyhedral stage. (*After Schwarze, Mycologia* **14**, 1922.)

Asexual Spores.—The young sporangiophores, especially towards the free ends, are filled with densely granular protoplasm containing many nuclei.

As the sporangiophore reaches its full length it begins to swell out at the tip into a tiny round body, the future sporangium. The contents of this are at first evenly distributed, being equally dense in the center and at the periphery, but before it has reached half its final size the protoplasm begins to be decidedly dense towards the sporangium wall, while in the center it is of a much looser structure (Swingle, 1903).

Numerous minute nuclei are scattered through both central and peripheral protoplasm, but they are more abundant in the peripheral portion. The central protoplasm is of a loose spongy texture with many large vacuoles, while the peripheral portion is dense and has a few very minute vacuoles. As growth continues the peripheral portion becomes more sharply marked off from the central vacuolated portion and finally a cell wall is laid down which separates the external *sporangium* or sporogenous portion from the central portion, which now constitutes the so-called *columella*. While the columella wall is being formed, changes are taking place in the dense peripheral protoplasm. Surface furrows or clefts appear which cut progressively inward, and gradually the whole of

the dense sporogenous protoplasm is cut up into angular masses of variable sizes containing two to six nuclei. These masses soon round off, and each forms a surrounding spore wall. During the process of maturing, the spores excrete a homogeneous slime which fills the intersporal spaces. The spores are set free by the breaking of the sporangium wall, but there is no explosive mechanism. The remnant of the sporangium wall may persist around the base of the columella as the so-called *collar* and separate the columella from the expanded end, or apophysis, of the supporting hypha. Some of the spores may remain for a time sticking to the columella, but they are ultimately scattered, leaving nothing but the old columella, which may finally become everted.

The sporangia are 100 to 350μ broad, snowy white when young, but black when old. The cell walls of sporangiophores and of root hyphæ are hyaline at first but become brown or brownish black with age. The spores are without any definite arrangement in the sporangium, variable in size, subglobular or broadly oval, generally longer than broad, 6 to 17μ in diameter, frequently with one or two blunt corners, and the external pale-gray wall marked by fine lines.

Sexual Spores.—*Rhizopus nigricans* is a heterothallic species, that is, certain mycelia are physiologically different from others, although identical in structure. Since they show no morphological sexual differences they may be designated as the plus (+) and minus (−) strains. When the mycelium of a plus strain mingles with that of a minus strain, sexual spores, or *zygospores*, are formed in the manner described (p. 490). In the fusion of the two gametes the nuclei are supposed to fuse in pairs. The mature zygospores are dark brown, globular or subglobular and 160 to 220μ in diameter, with external wall covered with hemispheric warts or projections.

THE SOFT ROT AND RING ROT OF THE SWEET POTATO

The soft rot and ring rot of the sweet potato are but different phases of the same trouble caused by *Rhizopus nigricans*, and not two distinct diseases, as was at one time supposed. The former is also called mush rot, vinegar rot or leak, while there are two phases of the latter called soft ring rot and dry ring rot.

History.—The first mention of soft rot of sweet potatoes as due to *R. nigricans* was by Halsted (1890). At the same time he described the ring rot, but attributed it to an entirely different organism, *Nectria ipomœæ* Halst., his diagnosis being based on the occurrence of the pinkish fruits of this fungus on the ring lesions. Taubenhaus (1914) studied both soft and ring rot and showed that the latter is caused by the same organism as soft rot (*R. nigricans*) and that *N. ipomœæ* Halst., as noted by Halsted, was probably only a saprophytic invader. The disease was further considered by Taubenhaus and Manns (1915) and also by Harter, Weimer and Adams (1918) in the general study of sweet-potato storage rots. Harter and Weimer have since published numerous papers dealing with various phases of *R. nigricans* and other species of Rhizopus which attack sweet potatoes in storage.

Symptoms and Effects.—Roots affected by *soft rot* become very soft and water soaked, and a clear liquid oozes out if the rotted tissue is broken. The rotted tissue is not changed in color at first, but later becomes a cinnamon or chocolate brown. "It has a characteristic wild-yeast odor at first, followed by a wild-rose to rose-geranium odor later." Pressure in the bins causes the breaking of the skin and the watery fluid may leak out, making adjacent roots wet, thus indicating the presence of the trouble. Nothing is seen of the aerial sporangiophores of the fungus unless the rotting potatoes are exposed to a very moist atmosphere or are broken open. Under normal storage conditions the humidity is not sufficient to make the fungus fruit unless the rotted tissue is exposed by a break or crack. At such points the characteristic sporangiophores will frequently develop in large numbers. With the evaporation of moisture the potato dries up, finally becomes mummified and in this final stage is frequently referred to as dry rot. Rotted potatoes may become mummified without the fungus ever appearing on the surface.

In ring rot the infection starts at one or more places between the two ends and the lesions advance around the potato to make bands or rings of rotted tissue, which by drying and shrinkage become more or less depressed. These rings may be 1 or 2 inches wide and the affected tissue extend ½ inch in depth or entirely through the root. The rotted tissue of a ring is soft at first, essentially similar to the tissue of the spreading type of soft rot, but when the fungus ceases to advance the affected tissue dries out and the dry rings are the final result.

While both soft rot and ring rots are primarily troubles which develop in storage, they are not confined to the harvested crop. They may appear in the field previous to digging time, and are most prevalent in low, moist areas, in delayed harvesting or when the roots have been injured in cutting the vines. Soft rot may also cause trouble to the sets in the hot bed, rotting the seed pieces and injuring the young sprouts.

Sweet potatoes suffer very heavily from storage rots, according to some estimates 30 per cent of the stored crop being lost. It has been estimated that of this loss nearly 20 per cent can be attributed to the ravages of *R. nigricans* or other Rhizopus species which behave in the same way. This condition seems to prevail from New Jersey to Alabama. The trouble still continues to take a heavy toll after the potatoes have been shipped to northern markets.

Etiology.—Taubenhaus and others have proved by pure culture inoculations that *R. nigricans* Ehr., unaided by any other fungi, can cause the symptoms and effects described. The fungus gains an entrance very largely through mechanical injuries of some kind. It is especially during the period of sweating that infections are likely to occur, and poorly ventilated storage houses greatly favor the trouble, due to the slow evaporation of the moisture. The rapidity of the advance of the rot

varies with conditions, but at room temperature a potato may be completely rotted in 4 to 6 days.

R. nigricans is a fungus of very general prevalence. The spores may be found almost anywhere in dust and dirt. They are present in the field and in the storage house. Even though the storage house is free from them at the beginning of the season, they will doubtless be brought in with the harvested crop. Some roots which were infected in the field will soon produce spores in the storage room and, as the rot spreads, more and more spores are prevalent, and, being resistant to desiccation, viable spores are ready to settle into any crack or bruise and start infections wherever sufficient moisture for their germination is available. The mycelium in the rotted roots is rather short lived. Taubenhaus (1914) has shown that it is generally dead after 12 to 15 days, and that various saprophytes then follow in succession on the same substratum. This will explain the early errors concerning the cause of ring rot. *R. nigricans* is the most common species of Rhizopus found on sweet potatoes, but a number of other species are able to produce a similar rot. Harter *et al.* (1921) have determined that eight other species are parasitic on sweet potatoes and that the different species can be roughly grouped into high, intermediate and low temperature forms. The high-temperature forms thrive best at temperatures varying from 20 to 40°C.; the intermediates, at temperatures varying from 20 to 35°C.; and the low-temperature forms, at temperatures ranging from 15 to 20°C. *R. nigricans* belongs to the low-temperature group, and is more favored by the temperatures at which sweet potatoes are ordinarily held. Rhizopus species cause rot by the secretion of the enzyme, pectinase, which dissolves the middle lamellæ of the cells and so causes their separation.

Varietal Susceptibility.—It had been frequently noted that some varieties of sweet potatoes are more susceptible to Rhizopus rot than others. This had been based largely on field observations until the work of Harter and Weimer (1921). Their tests of 16 commercial varieties showed that they could be divided roughly into three groups: (1) *very susceptible*, including Gold Skin, Little Stem Jersey, Early Carolina, Florida, Red Brazil, Haiti, Yellow Belmont and Dooley (100 per cent); (2) *intermediate*, including Porto Rico, Big Stem Jersey, Triumph, Pierson, Florida and Dahomey; (3) *quite resistant*, Nancy Hall and Southern Queen. These tests were based on the percentage of infection and on the rapidity of advance of the rot.

Control.—The prevention of Rhizopus rots of sweet potatoes is inseparably connected with the control of storage diseases in general, and requires special care in harvesting, curing and storage. Important features of harvesting are: (1) Dig only when roots are well matured; (2) avoid frost injury before or after digging; (3) dig in warm, dry weather rather than in wet periods; (4) do not store wet potatoes in great bulk;

(5) avoid long exposure to hot suns; (6) dig only what can be dried and picked up before night; (7) dig and handle with extreme care to prevent cutting or bruising.

The first 10 days to 2 weeks of the storage period is marked by the giving off of much moisture, amounting to 6 to 8 per cent of the original weight of the roots. Prevention of rot is dependent on producing as rapid an evaporation of this moisture as possible, as thoroughly dry surfaces prevent Rhizopus or other spores from germinating. The potatoes must, therefore, be cured for 10 to 18 days in a well-ventilated room with a temperature of 75 to 80°F. Following the curing period the temperature should be held at as near 55°F. (50 to 60°F.) as possible and the humidity should be held between 40 to 70, but below the higher figure, as that is the danger point.

The disinfection of seed roots with mercuric chloride is quite generally recommended. A similar reduction in strength occurs as in its use for the treatment of Irish potatoes, so if used continuously the treating solution soon becomes too weak to be effective. The present recommendation is based on the report of Weimer (1921). Dip in the bushel hampers or wooden crates in a solution of mercuric chloride, 1 ounce to 8 gallons of water, for 10 minutes and then spread out to dry at once. After treating 10 bushels, add $\frac{2}{5}$ to $\frac{1}{2}$ ounce of mercuric chloride and restore solution to the original volume. Discard the solution after treating 50 bushels.

LEAK OF STRAWBERRIES

Special attention has been given in recent years to the leak or soft, watery rot of strawberries, due to *R. nigricans*. It was reported as an important transportation trouble of strawberries by F. L. Stevens (1914), and later (1916) he stated that it is responsible for most of the losses to shipped berries. The rotting of strawberries in transit from southern states has been studied in some detail by N. E. Stevens and co-workers (1916 and later). It has been shown that Botrytis and Rhizopus species are the most important causes of rotting of strawberries, although there are many other fungi which may play a part. Botrytis is the cause of a rotting in the field previous to picking, while Rhizopus works largely on the harvested crop, but sometimes in the field (Melchers, 1921). The former does not produce a wet rot, but transforms the affected berries into hard, shriveled mummies. Rhizopus rots are the most frequent and destructive on berries shipped from the southern states, while in the New England states and in California, Botrytis is more frequently the cause of trouble.

Symptoms and Effects.—The berries rotted by Rhizopus soon become flattened, with considerable loss of juice, hence the appropriate name of "leak." The evident "whiskers" or aerial hyphæ which appear on

the rotted berries are an external indicator that the berries are already pervaded by the mycelium of the fungus. Shipments from the extreme South to northern markets are sometimes on the road for a week or more, and even fruit which appeared firm and sound at the time of picking may reach its destination in a badly rotted condition.

Etiological Relations.—Stevens has shown that Rhizopus alone, without the aid of any other fungi, may give rise to all the characteristic symptoms and effects of leak. It is not necessary for Botrytis or some other fungi to pave the way for its entrance. It does not seem to be able to enter the unbroken epidermis, but in soft fruits like strawberries it is difficult to handle them without causing bruises which are sufficiently numerous to serve for entrance. The hyphæ of the Rhizopus enter between the cells and ramify in the intercellular spaces, rarely if ever entering the cells. They are confined largely to the periphery of the fruit,

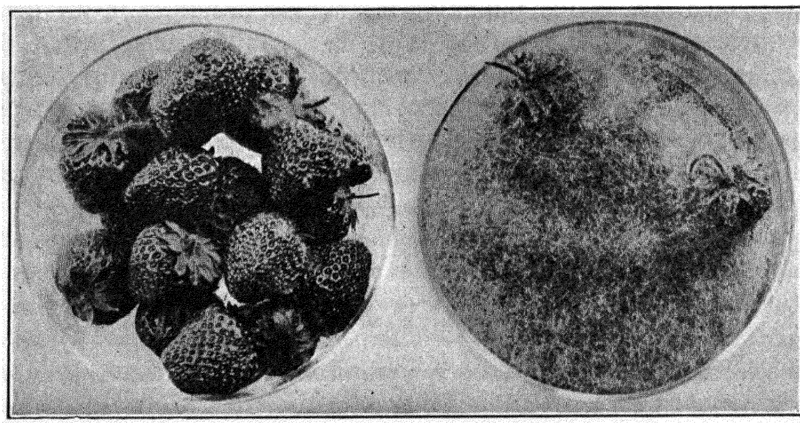

FIG. 141.—Strawberries slightly and severely attacked by *Rhizopus nigricans*, the cause of leak. Berries held for 48 hours in moist chambers.

but may penetrate deeper if rotting takes place under rather dry conditions. While the fungus dissolves the middle lamellæ, as noted for the sweet potato, it apparently has an effect on the protoplasm which changes its permeability and so allows the cell sap to escape into the intercellular spaces.

It has been shown that the temperature relations and mechanical injuries are the most important factors influencing the amount and severity of the Rhizopus rot. The rot develops rapidly at temperatures between 15 and 20°C. and slowly or not at all at 10°C. or lower. It has been noted that berries picked and packed when wet keep better than those picked dry, or those picked in the cool of the morning keep better than those picked in the heat of the day. If strawberries must be washed to remove sand they should be packed and shipped wet without any attempt at drying, but washing of berries that have been picked dry is not to be recommended (Stevens and Chivers, 1919). These relations were

at first attributed to the effect of the evaporation in lowering the temperature and so making conditions less favorable for the growth of Rhizopus, but the recent results of Hawkins and Sands (1920), which show that lowering of temperature increases the resistance of the epidermis to puncture or mechanical injury, are especially significant. Lowered temperatures mean less mechanical injury, consequently less opportunity for the entrance of Rhizopus.

Control or Prevention.—Careful attention should be given to sanitation in the field and in the packing house to remove sources of infection. Packing house and tables should be kept clean, and cull fruit should not be allowed to accumulate where it will be producing crops of spores, but should be destroyed. The berries should be handled as little as possible, and, when handled, the utmost care should be taken to prevent bruising. Advantage should be taken of the known facts concerning the temperature relations of the rot. Berries for shipment should be kept as cool as possible from the time they are picked, and should be refrigerated as early as possible. Delay between picking and refrigeration is sometimes disastrous. Even though the berries may be contaminated with Rhizopus spores, with proper handling and with adequate refrigeration in transit (10°C.) they should reach the market in good condition. "Pony" refrigerators have made long-distance express shipments possible.

OCCURRENCE OF RHIZOPUS ON OTHER HOSTS

The two crops considered, sweet potatoes and strawberries, suffer more generally and more severely from Rhizopus rots than any others, but various vegetables and fruits suffer at times from soft rots or leak. It has been shown that *Rhizopus nigricans* is one of the causal agents of leak or melters of the Irish potato (Orton, 1909; Hawkins, 1916), although another fungus (*Pythium debaryanum*) is more frequently associated with this trouble. Apples, pears or quinces may be attacked, while plums, peaches and cherries also suffer. The writer observed one case of severe Rhizopus leak in sweet cherries which were held several days in common storage before shipment. Currants, raspberries, blackberries and mulberries have all been affected, in fact it seems that no soft fruits are exempt. Rhizopus rot of tomatoes has been reported from England (Wormald, 1912) and Germany (Behrens, 1898), and has been occasionally noted in the United States. A soft rot of the fig in Louisiana has been described by Edgerton (1911) as due to *R. nigricans*.

The trouble occurs chiefly during rainy spells in the summer when the fruit is ripening. The fruit sours, becomes soft and rotten and, finally, generally falls to the ground. At the time the fruit falls it is generally so soft that it goes all to pieces when it strikes the ground.

Rhizopus has long been known to cause injury to germinating seeds (Muth, 1908), but Adams and Russell have recently (1920) studied the

Rhizopus infection of corn on the germinator and have shown that the fungus penetrates the scutellum, causing a "retarded growth, underdeveloped shoot and subnormal color of the leaves."

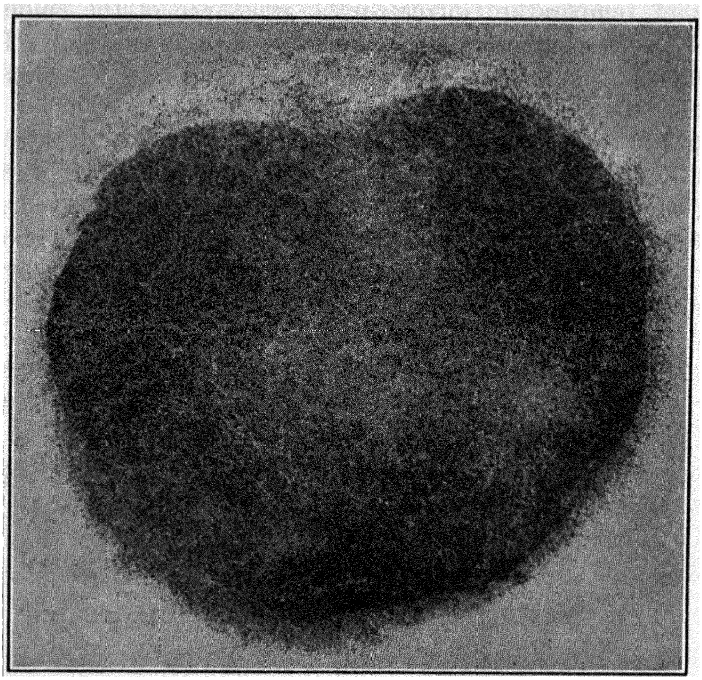

FIG. 142.—Development of sporangiophores from cut surface of an apple rotted by *Rhizopus nigricans*.

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BLOSSOM BLAST AND FRUIT ROT OF THE SQUASH

Choanephora cucurbitarum (B. & Rav.) Thax.

This disease is characterized by a blighting of the blossoms followed by a rotting of the young fruit. The fungus was first described by Berkeley from decaying squashes from South Carolina (1875). It was reported from Brazil by Möller (1909), who found it on hibiscus flowers. Clinton reported it on squash flowers in Connecticut in 1902–1903, and it was also studied by Thaxter (1903). Both writers considered it parasitic on the squash. A recent study is by Wolf (1917), who attached importance to it as a disease of the squash in North Carolina, where it was found "most destructive on the 'pattypan' types of summer squashes commonly known as cymlings." It appears to be epiphytotic only under conditions of high humidity and excessive rainfall. It was noted by the writer in Texas on summer squashes in 1911. More recently it has been reported on chillies (Dastur, 1920) and as the cause of a disease of amaranth (Palm and Jochems, 1924).

Symptoms and Effects.—The flowers or young fruits become covered with a luxuriant growth of conidiophores. They are first evident the day following the opening of the flowers, and at this time the conidial heads are white. The fructifications soon change to brown and finally to purplish black, and then have a peculiar metallic luster. The attacked tissues turn brown and become soft and apparently water soaked. Both staminate and pistillate flowers may be affected, and in the former the pedicel may also be involved. In pistillate flowers the fungus advances from the corolla into the young squash and a soft, wet rot is the result. In the most severe cases all of the flowers of a vine may be blighted or the fruits may be blighted when very young or rotted after they are partially grown.

Etiology.—This trouble has been attributed to *Choanephora cucurbitarum* (B. & Rav.) Thax., the causal relation being based largely on association rather than on inoculations with pure cultures. Infections are supposed to take place through the corolla. Under normal conditions the faded, shriveled corolla persists for some time, and under very humid conditions this offers a very suitable substratum in which the fungus can establish itself, and from which the advance can be made into the young delicate tissues of the developing fruits. The mycelium is both intercellular and intracellular, and produces no evidence of necrotic changes beyond the tissues actually invaded. The same fungus has been found on the faded flowers of cucumber (*Cucumis sativus*), rose of Sharon (*Hibiscus syriacus*), scarlet hibiscus (*H. coccineus*), okra (*H. esculentus*) and cotton (*Gossypium herbaceum*).

The simple erect *conidiophores* which are produced in such profusion on the host parts are expanded at the tips into capitate enlargements.

Each head, or capitulum, produces a few to a dozen or more cylindrical branches, each one of which also forms a globular head, and from these the conidia are formed by a budding process. The young heads are white, but they soon darken, and with the closely clustered conidia present a superficial appearance not unlike an Aspergillus. The conidia are oval to elliptical, longitudinally striate, light to reddish brown, 15 to 25 by 7.5 to 11μ, and each is provided with a delicate hyaline appendage, a portion of the sterigma on which it was borne. The sporangiophores have been

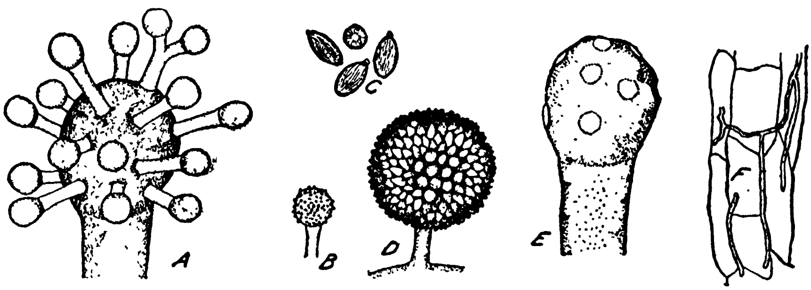

FIG. 143.—*Choanephora cucurbitarum*. *A*, immature conidiophore with ramuli developing on the primary vesicle; *B*, bud-like processes, the beginning of conidial formation on the capitellum; *C*, conidia; *D*, mature capitulum covered with a layer of conidia; *E*, primary vesicle from which ramuli have been detached; *F*, mycelium in tissue of squash-flower pedicle. (*After Wolf*, *Jour. Agr. Res.* **8**, 1917.)

studied mostly from cultures, since they seem to be rarely produced on the host. The sporangia appear as pendant, white, globular structures, which become black at maturity and vary from 35 to 100μ in diameter. The sporangiospores are ovoidal or elongated, sometimes inequilateral, colored like the conidia and 18 to 30 by 10 to 15μ. The walls are smooth, but provided with either lateral or terminal tufts of hair-like appendages. The species is heterothallic according to Blakeslee, and zygospores are formed in the way outlined for *Rhizopus nigricans*. They vary from 50 to 90μ in diameter. The spores of the fungus are disseminated by the wind, but it seems probable that insects are also very important carriers. Bees and the common striped and spotted cucumber beetles appear to be important agents, since Wolf has shown that the spores of the pathogene can be easily recovered by making microscopic tests of water in which these insects have been washed.

Control.—No data are available on control. Since many more blossoms are formed than can set fruit, it seems probable that special control measures will not be necessary under average conditions.

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IMPORTANT DISEASES DUE TO MUCORALES

Soft and ring rot of sweet potato (*Rhizopus* spp.).—(See special treatment, p. 494.)

Leak and rot (*Rhizopus nigricans* Ehr.).—Causes a rot of various fruits, and is one of the causal agents of leak of Irish potatoes (see p. 450).

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CHAPTER XIX

DISEASES DUE TO LEAF CURLS AND RELATED FUNGI

EXOASCACEÆ

The leaf curls and related fungi are obligate parasites belonging to the primitive ascomycetous family, the Exoascaceæ. They attack mainly woody species, but one genus contains species which parasitize ferns. The principal hosts of economic importance are either forest or fruit trees. Among important forest-tree hosts the following may be noted: birch (Betula), alder (Alnus), oak (Quercus), poplar (Populus), iron wood (Carpinus), hop hornbean (Ostrya), and hawthorn (Cratægus). Wild and cultivated species of Prunus (especially peaches, plums and cherries) and some Pyrus species are the noteworthy hosts among fruit trees.

General Characters.—Both vegetative and reproductive characters of the family are so distinctive as to permit a ready recognition. The important features are (1) an abundant intercellular (with a single exception), septate mycelium of very irregular cells, which vary greatly in length and diameter; (2) the production of four- to eight-spored asci in extensive palisade-like layers, which originate from the intercellular mycelium, either beneath the cuticle or under the epidermal cells (in the epidermal cells in species of Taphrinopsis on Pteris); and (3) the tendency for ascospores to bud either in the ascus or after their discharge. In the case of budding of ascospores previous to their liberation, asci may appear to bear many ascospores instead of the limited number characteristic of most ascomycetes.

It has recently been shown for several species that the bud-conidia originating from the ascospores represent plus and minus types capable of fusion, certain ascospores yielding only plus and others only minus conidia. In the fusion a conjugation canal is formed, and the content of the one conidium passes into the other, after which an infection thread may be formed. The extent of this behavior is uncertain, but it seems probable that further studies will show it to be common.

The effects of leaf-curl infections vary with the different species and hosts and may be summarized as follows: (1) Leaf spots with slight or no hypertrophy of the tissue; (2) discoloration and deformation of leaves or of extensive leaf areas, which may be blistered, crinkled, curled, arched or puckered with increased thickness; (3) deformation of flower parts or fruits; (4) slight shoot deformation with discolored and malformed leaves; and (5) pronounced witches' brooms bearing malformed leaves. In

most cases there is more or less hypertrophy, frequently accompanied by hyperplasia. The varying responses result from enlargement of cells, increase in number of cells, forcing of dormant buds, modified geotropism of shoots, premature or delayed development and may cause the death and fall of leaves, the suppression of flowering or fruiting or the killing back of young twigs or extensive witches' brooms. In some species the infections are annual, while in others, like the broom-forming species, the mycelium is perennial, persisting for years in the affected twigs.

The most characteristic feature of the family is the spreading out of the ascus layer over the surface of localized leaf spots or of hypertrophied plant organs, especially leaves or carpels, rather than the organization of specialized ascus fruits.

The Genus Taphrina.—Formerly, two important genera of Exoascaceæ were recognized: Exoascus and Taphrina, the former with four- to eight-spored asci, the latter with many-spored asci. Since the many-spored asci were shown to be the result of budding, this separation of the two genera was abandoned, and when other distinguishing characters were not found, the two were united under the single genus, *Taphrina*, in three subgenera:

1. *Taphrinopsis*, with club-shaped asci narrowed at the base; parasitic on ferns.
2. *Eutaphrina*, with plump, cylindrical asci, more or less truncate at the apex; parasitic on Amentaceæ, especially birches, alders, oaks, etc.
3. *Euexoascus*, with club-shaped to narrowly cylindrical asci more or less rounded at the apex; parasitic on Rosaceæ, especially on species of Prunus and Pyrus.

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PEACH LEAF CURL

Taphrina deformans (Fcl.) Tul.

The peach and its derivatives are affected by a serious fungous disease, the most striking effect of which is the production of thickened curled leaves of yellowish or reddish tints instead of the normal color. On account of this characteristic effect, it has been called peach leaf curl, but less frequently curl, curly leaf or leaf blister (*Kräuselkrankheit*, German; *Cloque du pêcher*, French).

History.—The leaf curl of the peach was known in England at least as early as 1821, being described by an English gardener as a type of blight called blister and curl. It was years afterwards before the parasitic nature of the disease was recognized and the causal organism described by the English mycologist Berkeley in 1860. There are records of the occurrence of the disease in Australia as early as 1856. Various attempts have been made to determine the country of origin of peach leaf curl, but no positive information has been obtained. There are some indications that it came from China, the original home of the peach, since seedlings are generally very susceptible and the Chinese Saucer peach is lacking in resistance. The disease must have been well established in European countries by 1880, as it was given prominent consideration by scientists in Great Britain, Germany, France and Italy about that time and during the years immediately following.

While European workers understood something of the nature of the parasite and described the symptoms and effects of the disease, they were slow in devising any effective control measures. From the earliest work up until about 1896 they considered the parasite perennial in the twigs and buds of the host and recommended the destruction of diseased leaves and the cutting out of affected twigs as the only means of control. The first real progress in control was made by accident by California growers about 1880–1885, when they began using the lime, sulphur and salt solution for the control of San José scale. They found that their trees given this dormant spray previous to the opening of the buds were generally free from leaf curl. Experiments carried out by Benton in the orchard at the University of California in 1890 showed that winter treatment with copper sprays would also effectively control the

disease, that summer treatment was entirely ineffective and that infection of the spring growth by a perennial mycelium was the exception rather than the general rule. The copper treatment for leaf curl was independently discovered in Australia in 1891. Notwithstanding the success of Californian and Australian workers, the successful control of leaf curl in Europe was not recorded until 1898. Even workers in the eastern United States were slow to learn about or to accept the findings of their western neighbors. Successful control was recorded by Taft in Michigan by 1893, but several years later Duggar (1899) and Murrill (1900) published bulletins from Cornell University giving experiments on control which seemed to indicate that they were unfamiliar with the practices in California. A very exhaustive study of leaf curl was begun in 1893 by Pierce of the U. S. Department of Agriculture, culminating in the publication of a large bulletin in 1900. This work was largely responsible for our present practices in control, which have been modified only in minor details.

Geographic Distribution.—The leaf curl of the peach has become practically world wide and is known in all countries where peaches are grown, although it has not developed in certain peach-growing regions where climatic conditions are generally unfavorable. Previous to the discovery of the successful methods of control, it was considered the most important fungous disease of the peach tree, especially in the cooler climates. It has been destructive throughout Europe, parts of China and Japan, northern and southern Africa, Australia and to some extent in South America and New Zealand. In America the disease has been especially serious in the Pacific Coast section, including peach districts from California to British Columbia, west of the Cascades. The most important irrigated districts of central Washington have, however, remained free from the disease. East of the Rocky Mountains the disease has been most prevalent in the Great Lakes regions of Michigan, Ontario, Ohio, New York and adjacent territory. In the warm, semiarid regions of the Southwest it is either rare or entirely unknown.

Symptoms and Effects.—The leaf curl affects leaves, tender growing shoots and more rarely blossoms and fruits, but is most conspicuous from its leaf attacks. The disease is first in evidence very soon after the young leaves begin to unfold from the bud. The diseased leaves are either noticeably reddened or paler in color than normal and become much curled, puckered or distorted, at the same time being greatly increased in thickness, and of a firm or cartilaginous consistency. Any portion of a leaf or the entire leaf may be involved and all or only part of the leaves from a bud may be affected. Only a few leaves may be affected or the infections may be so numerous as to destroy almost the entire foliage. The bright color of the affected foliage is a striking feature of the disease, making the diseased trees especially conspicuous. As the season progresses, the red coloration becomes less pronounced, chlorophyll disappears almost entirely and a grayish bloom or powdery coating (the spores) appears over the upper surface of the affected portions of the leaves. As might be expected, the transpiration of affected leaves is increased. After this stage, the leaves gradually turn brown, wither and fall from the tree in the latter part of June or early in July, the length of time required for defoliation depending upon the climatic conditions which prevail, the process being hastened by hot, dry weather. Attacks of leaf curl may show all stages of severity from a few scattering infections to complete

defoliation. Following the loss of leaves, a new set will be produced from dormant buds.

The young terminal twigs may also be involved, being reduced in length, more or less swollen and pale green or yellow. In such cases the end of the shoot may be enlarged for a length of 4 or 5 inches. Such affected shoots generally produce nothing but curled leaves, and the majority die back, but in some the terminal bud may continue to grow and produce a healthy shoot, while the hypertrophic portion will remain as a rough cankered zone at its base, sometimes showing exudation of gum. This behavior of the disease is by no means general, but is probably responsible for the old belief that the parasite was perennial in the twigs.

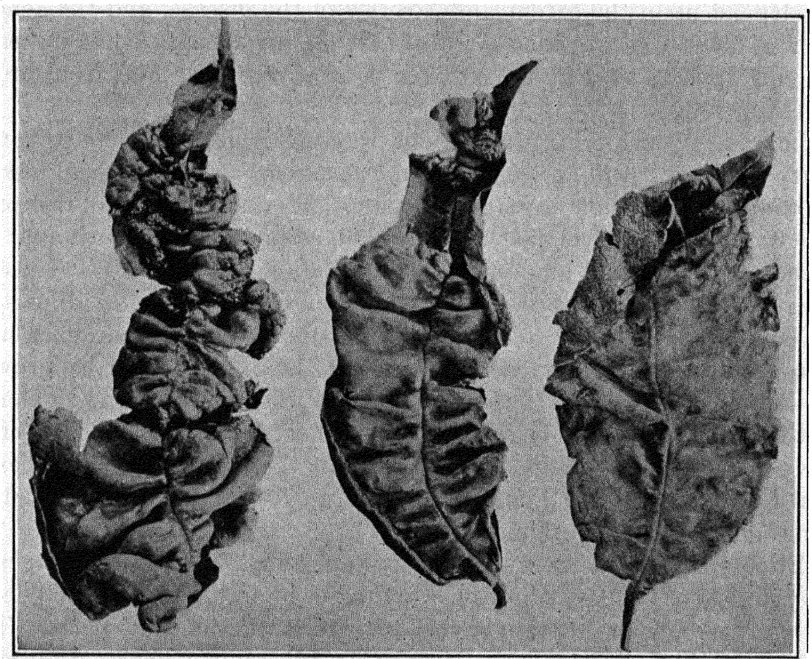

Fig. 144.—Peach leaves affected with leaf curl. (*Photograph by B. F. Dana.*)

In severe infections the blossoms and young fruits are affected, but these are blighted and fall from the tree very early; consequently this phase of the disease is frequently overlooked.

Young fruits when infected become much distorted, owing to the portions becoming greatly enlarged. Such fruits seldom remain long on the tree, as they become scabbed and cracked, and soon fall. Maturing fruits are commonly attacked—this phase of the disease being more common than is generally recognized—and as a result swollen, irregularly shaped areas, usually bright colored, appear on the surface. These areas are much wrinkled, and on peaches often appear as if polished, owing to the absence of those hairs which normally cover the surfaces (Cunningham, 1923).

The injury from leaf curl may be summarized as follows: (1) the loss of the foliage in the spring is a heavy drain on the vitality of the trees, since a new crop must be produced; (2) repeated loss of foliage through a period of years may so lower the vitality as to cause the death of trees; (3) lowered vitality may reduce or entirely prevent the setting of fruit buds for the next year; (4) lowered vitality and prolonged growth at the end of the season may make the trees more likely to winter kill; (5) blossoms and young fruits drop because of direct attacks; (6) fruits fail to set or to remain on the tree because of defoliation, even when direct invasion does not occur; (7) terminal twigs are killed back or buds are killed in nursery stock. The amount of injury suffered in a given case will depend upon the extent and severity of the disease, and the general vigor of the trees previous to and following an attack. In some cases leaf curl appears to pave the way for more severe infections from brown rot (Mix, 1930).

Losses.—Considering the various ways in which the disease causes injury to trees, it will be seen that an estimate of actual financial loss for the country as a whole is rather difficult. At a time when control practices were not well developed, Pierce (1900) estimated that the annual loss to the country would amount to $3,000,000 or more. This was a conservative estimate based on a detailed and extensive survey of all parts of the country. With the adoption of effective spraying, the losses have been greatly reduced in recent years, but there are still many growers who are either ignorant of the control practices or are willing to chance that the disease will not be serious. It is now nearly 25 years since effective control practices have been known, but the annual loss has recently been estimated by the Plant Disease Survey to amount to as high as 15 per cent in some sections.

Etiology.—Leaf curl is caused by an obligate parasite, *Taphrina deformans* (Fcl.) Tul., a primitive sac fungus, or ascomycete, which develops an intercellular, septate mycelium within the affected parts. This fungus was first described by Berkeley in 1857 as *Ascomyces deformans*, but was later named *Exoascus deformans* by Fuckel (1869). It was described by Tulasne as *Taphrina deformans* in 1866 and that name is employed by most current writers.

The mycelium of the fungus may be readily observed by making a dissection of affected tissue that has been treated for a few minutes in warm, dilute caustic potash, or carefully prepared sections may be used when detail of arrangement and relation to host cells are desired. The mycelium is found to be quite generally present in the intercellular spaces of hypertrophied leaves and in the cortex of swollen twigs. Pierce has recognized three types: (1) *Vegetative hyphæ*, the common type in the parenchyma tissue of leaves, leaf stalk or cortex of affected or distorted branches. This vegetative mycelium shows cells of very irregular form

and size, much curved and twisted and of varying diameter. (2) *Distributive hyphæ,* the form found close beneath the epidermal cells of diseased twigs and very abundant in the pith. They are composed of more elongated cells, of rather uniform diameter, and are arranged parallel to the stem axis. They are supposed to be concerned primarily with spreading the fungus in the branch. (3) *Fruiting hyphæ,* the kind developed between the cuticle and the epidermal walls of the upper epidermis. Vigorous, well-developed vegetative hyphæ fill the intercellular spaces just below the upper epidermis of the leaf, and branches from these penetrate between the epidermal cells and form an extensive layer of

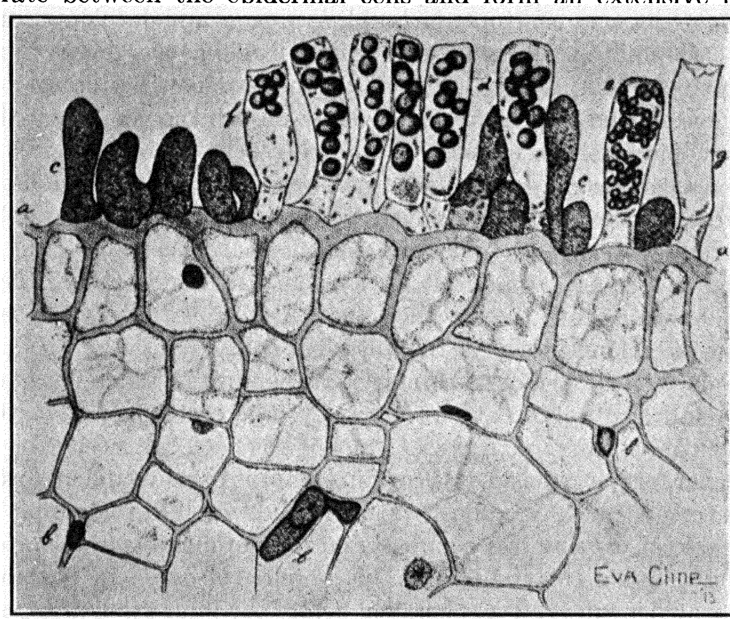

FIG. 145.—Section of a peach leaf affected with leaf curl. Asci containing ascospores are forming on the upper surface; *a*, cuticle of leaf; *b*, bits of mycelium of the fungus; *c*, young asci with spores not yet formed; *d*, spores just formed; *e*, spores being divided into smaller ones; *f*, spores discharging; *g*, empty ascus. (*After Swingle, Mont. Agr. Exp. Sta. Circ.* **37**, 1914.)

short, more rounded cells, the *ascogenous* cells, between the cuticle and the upper epidermal wall. These ascogenous hyphæ are filled with a dense granular content of nutritive material for the development of the asci. The asci are formed by upward elongations from the ascogenous cells, and a cross-wall is formed in each, leaving a stalk or foot portion and the terminal ascus. The subcutaneous and intercellular hyphæ are binucleate. Fusion takes place just prior to ascus formation. This upward growth of the developing asci raises the cuticle and this is either pierced or torn and disappears, leaving the asci exposed to the surface as a more or less continuous plush-like coating, which in section will appear as a palisade-like layer.

The asci are usually flattened or somewhat truncate at the free end, broader above than below, and vary from 25 to 44μ long by 8 to 12μ in diameter. Each ascus normally forms eight spores, but the number may vary from three to eight. The ascospores are oval to spherical, 6 to 9μ by 5 to 7μ, hyaline and surrounded by a firm but rather inconspicuous wall. They are forcibly expelled through an apical slit or rupture in the ascus and may accumulate on the surface of the leaf, giving the white or grayish powdery condition described in the consideration of symptoms. The ascospores may form buds, and these may separate as primary conidia either within the asci or after they have been set free. These primary conidia may continue the budding process and develop secondary conidia. Copulation of plus and minus conidia has been reported (Weiben, 1927; Mix, 1929). In this way the fungus will produce a slow-growing, yeast-like, delicately pink colony on solid media, consisting of budding conidia, short mycelia and resting cells (Mix, 1924; Martin, 1925). Ascospores may also germinate direct by the formation of stocky germ tubes. The exact part played by these two types of germination in the production of new infections is somewhat uncertain. The ascospores or the conidia produced from them are borne away from the diseased leaves before they wither and fall and nothing more is seen until new infections appear in the following spring.

The place of sojourn of the fungus and the form in which it passes the summer, fall and winter are uncertain, but it seems probable that it hibernates on various tree parts to which it has been carried by the wind. In whatever form it persists, it is certain that it is present in such form and place as to cause new infections when the buds swell in the spring but is also capable of causing less common and less serious infections after the unfolding of buds (Mix, 1925). This opinion is based in part on the following evidence: (1) Trees sprayed before the buds swell in the spring show little or no infection, while unsprayed trees under exactly the same conditions develop the disease; (2) the disease generally appears first on leaves just as they are unfolding from the bud; (3) spraying after the buds have swollen does not prevent the disease.

From the recent careful work of Mix (1924), the following features are worthy of consideration as bearing upon the life history of the leaf-curl fungus: (1) The conidia produced by the budding of the ascospores can grow saprophytically in cultures and may perhaps do so in nature; (2) the conidia are able to withstand desiccation sufficiently long to enable them to survive until infection occurs in the spring; and (3) the conidia can and do infect young unfolding leaves as shown by inoculation experiments. Whether the thick-walled resting cells which appear in cultures play a part in nature or not is uncertain.

It was formerly supposed that the fungus was perennial in the twigs and invaded the opening buds, without the necessity of new infections

taking place. While this may occasionally be true, the success of spraying in controlling the disease, by preventing 90 to 98 per cent of the infections, shows that perennial mycelium plays a very minor part in the life of the fungus. Also, the prevalence of the disease only during cold, wet springs is in opposition to any general persistence of a perennial mycelium.

Pathological Anatomy.—The first effect of the leaf-curl mycelium is to irritate the host cells and stimulate them to an abnormal activity. In the infected leaf, the cells are increased in size and number and marked changes in form and structure occur. This increase in size and number of cells is most marked in the palisade parenchyma, and the affected cells suffer almost a complete loss of chlorophyll. The increase of cells on

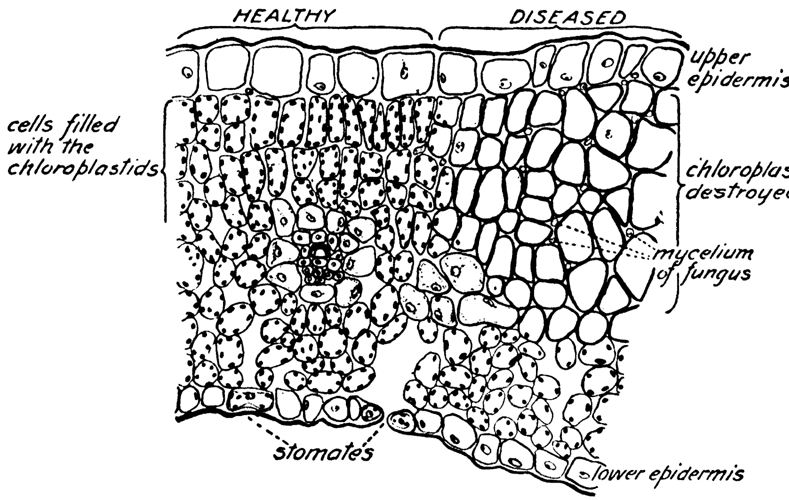

FIG. 146.—Cross-section through a peach leaf affected with leaf curl along the line of diseased and healthy tissue. (*After Wallace and Whetzel, Cornell Univ. Agr. Exp. Sta. Bul.* **276.**)

either side of the midrib causes a pronounced gathering or cross-wrinkling, with the midrib acting like a puckering string. At the same time the affected leaves generally become more or less concave below and convex above, since the palisade parenchyma suffers more hyperphasia than the spongy parenchyma. The mycelium is entirely intercellular, and produces no haustoria, or sucking organs, to penetrate the host cells, but does come into very close physical relation to them.

In affected shoots there is a great increase in the number and size of the cells of the cortical parenchyma. The cells are more angular than normal and show variation in the thickness of the walls. The cortex may be eight to ten times as thick as normal. Such affected shoots generally show greatly shortened internodes, with the enlarged leaves crowded to give a plumed, tufted or rosette appearance.

Predisposing Factors.—It is perhaps fortunate that leaf curl requires certain conditions for its development that are not offered every season

in a given environment. It is a notable fact that the disease is epiphytotic at certain periods, while during another season it may be almost entirely absent. It has been generally observed that curl is favored by cold, wet weather when the leaves are opening from the bud, and it has been shown by careful studies of epiphytotics that their occurrence is dependent upon the weather conditions. Low temperature and wet weather at the critical time, when the leaves are unfolding, the time when the majority of infections take place, are believed to favor the disease for the following reasons: (1) Transpiration is checked out of all proportion to root absorption, and the leaf tissue becomes gorged or distended with water, making it very susceptible to the fungus; (2) the growth of the host is retarded, while at the same time the moisture and temperature conditions are favorable for the germination and growth of the fungus. In some regions the absence of leaf curl is explained largely by the failure to have proper conditions of either moisture or temperature at the normal infection period.

On the other hand, the general absence of the disease from certain regions like the peach-growing sections of Texas and the Southwest may be explained on another basis. It seems probable that the conidia would not be able to withstand the devitalizing temperatures which prevail during the summer months. This conclusion is suggested by the fact that conidial cultures are "completely devitalized when kept for a few days at 30°C." (Mix, 1924).

The above consideration will perhaps make it clear why leaf curl is more likely to be severe in the neighborhood of large bodies of water—for example, near lake shores or along river valleys. In such localities there is a greater humidity of the air and the temperature factor in the early spring is more likely to be favorable. Rainfall alone is a minor factor, as may be noted in the Pacific Northwest, where the disease is severe in the region of heavy rainfall west of the Cascades and also severe in certain localities east of the Cascades in which the rainfall is light. Regions of heavy dews, but with light rainfall and early warm spring weather, are unfavorable to the disease.

Host Relationships.—Leaf curl is distinctively a disease of the peach or its derivatives, such as the nectarine and the peach-almond. Its occurrence on the almond may be considered very exceptional. Among varieties of peaches great variations are shown in susceptibility to the disease. It seems to be quite generally conceded that seedlings are more susceptible than budded trees, although many budded trees show very high susceptibility.

Pierce made a very exhaustive study of nearly 200 varieties and came to the conclusion that varieties which are reported resistant in one environment may be susceptible when grown under other conditions, and this is substantiated by reports from other countries. Some of the best com-

mercial varieties seem to be very susceptible—for example, the Elberta, a general favorite, and the Lovell, a favorite in California. Since it is possible "successfully to grow the most susceptible varieties in the most unfavorable situation" (Pierce) by following the accepted methods of control, growers should give first consideration to the selection of varieties otherwise suitable to their environment or to their trade and only secondary consideration to their behavior with reference to leaf curl. Because of variation in resistance, selection should be made on the basis of regional experience, rather than on reputed resistance in distant sections.

Control.—Previous to our knowledge of the real habits of the leaf-curl fungus, recommendations for control were made which we now know are entirely ineffective (see History). It has been shown that the leaf-curl fungus persists on the trees in some form during the dormant period and that it can be reached and killed by sprays applied at that time. It has repeatedly been demonstrated that the disease can be effectively controlled by a single application of fungicide—Bordeaux, copper sulphate or lime sulphur—previous to the swelling of the buds in the spring. The question may very properly be asked as to what constitutes effective control. The results obtained by a number of experimenters may be cited:

Experimenter	Unsprayed, per cent leaves curled	Sprayed, per cent leaves curled
Murrill, 1900	23	0–2.5
Wallace and Whetzel, 1909	34.3	2.3–6.1
Wallace and Whetzel, 1909	41.3	5.3–8.3
Wallace and Whetzel, 1909	58.9	0.9
Reddick and Toan	16.87	0.016–0.05

Numerous reports from practical orchardists claim almost no leaf curl on sprayed trees and heavy infection on unsprayed trees. No carefully planned experiments have given failure, but some orchardists have claimed that spraying did not control the disease. These failures are undoubtedly due to lack of attention to the factors which make for success. These are: (1) the time of application of the fungicide; (2) thoroughness of application; (3) the fungicide employed. Of these three factors, the first two seem more likely to be the cause of failures, since good results have been obtained with copper sulphate, 2 pounds to 50 gallons of water, different strengths of Bordeaux, from 3–3–50 to 6–6–50, and commercial lime sulphur (Baumé test 32°) 1–9 to 1–15. Promising results have recently been obtained by the use of soluble sulphur dusts (Massey and Fitch, 1923).

To be successful, the fungicide selected must be applied before the buds begin to swell in the spring, as the fungus must be killed before any opportunities are afforded for infection. It was formerly thought that

the successful treatments would be obtained only when spraying was carried out during 1 to 3 weeks previous to the opening of the buds, but tests carried out in New York have given successful control when the trees were sprayed in either late fall or winter. Complete covering of the buds is also essential to success; hence best results will be obtained with high pressure in dry, calm weather.

It should be noted that neither plain copper sulphate nor Bordeaux are of any value in the control of San José scale; hence if these insects are present, it will be advisable to use lime sulphur rather than one of the copper fungicides. By using lime sulphur 1–8 as a dormant spray, San José scale will be controlled and leaf curl will be reduced to a negligible amount, by the single application.

The final recommendations for control are as follows: (1) Spray *once*, either in the late fall, early winter or in the spring *before the buds start to expand*, using the fungicide that is best suited to the orchard conditions which prevail; (2) spray thoroughly, that is, cover every bud with the fungicide; (3) trees defoliated by leaf curl should be given the best of care and culture in order that they may overcome the drain occasioned by the attack. The protective spray should certainly be given the season following severe injury.

Do not spray after the leaves have opened, as such an operation has little or no effect in preventing leaf curl and may cause injury to the healthy foliage.

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IMPORTANT DISEASES DUE TO LEAF CURLS AND RELATED FUNGI

TAPHRINOPSIS

Leaf blister (*Taphrina filicinia* Rostr.).—On fronds of *Aspidium spinulosum* and *Dryopteris acrostichoides*.

Witches' broom of Pteris (*Taphrina laurencia* Gies.).—Causes profusely branched, foliaceons broom-like clusters on the fronds of various species of Pteris in the Orient. LAUBERT, R.: *In* Sorauer's Handbuch (5te Auf.) **2**: 465–466. 1928.

EUTAPHRINA

Poplar-catkin disease (*T. johansoni* Sadeb.).

Poplar yellow-leaf blister (*Taphrina aurea* (Pers.) Fries).

Birch red-leaf blister (*Taphrina bacteriosperma* Joh. and *T. carnea* Joh.).

Birch yellow-leaf blister (*Taphrina flava* Farl. and *T. betulæ* (Fckl.) Joh.).

Birch witches' broom (*Taphrina turgida* (Rostr.) Sadeb. and *T. betulina* Rostr.).

Alder-catkin disease (*Taphrina alni incanæ* (Kühn) Magn. and *T. amentorum* Sadeb.).

Alder leaf blister (*Taphrina tosquinetii* (West.) Magn. and *T. sadebeckii* Joh.).

Alder witches' broom (*Taphrina epiphylla* Sadeb.).

Ironwood leaf curl (*Taphrina australis* (Atk.) Gies.).

Ironwood witches' broom (*Taphrina carpini* Rostr.). LAUBERT, R.: *In* Sorauer's Handbuch (5te Auf.) **2**: 467–481. 1928.

EUEXOASCUS

Plum pockets (*Taphrina pruni* (Fcl.) Tul. and *T. communis* (Sad.) Gies.).—Perennial in wild and cultivated plums causing the fruits to become greatly inflated and hollow. Also called "bladder plums" and "fools." HESLER, L. R. AND WHETZEL, H. H.: Manual of Fruit Diseases, pp. 373–377. The Macmillan Company. 1917. SWINGLE, D. B. AND MORRIS, H. E.: Plum pocket and leaf gall on American plums. *Mont. Agr. Exp. Sta. Bul.* **123**: 167–188. 1918. STAKEMAN, E. C. AND TOLAAS, A. G.: The control of brown rot of plums and plum pockets. *Minn. Hort.* **46**: 5 pp. 1918. CUNNINGHAM, G. H.: Leaf curl, bladder plum and cherry curl. *New Zeal. Jour. Agr.* **26**: 85–97. 1923.

Witches' broom of cherry (*Taphrina cerasi* (Fcl.) Sad.).—HESLER, L. R. AND WHETZEL, H. H.: Manual of Fruit Diseases, pp. 188–191. The Macmillan Company, 1917. SCHMITZ, HENRY: Some observations on witches' broom of cherries. *The Plant World* **19**: 239–242. 1916.

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Peach leaf curl (*Taphrina deformans* (Fcl.) Tul.).—(See special treatment.)

Pear leaf blister (*Taphrina bullata* (Fcl.) Tul.).—Also affects quinces. BRITON-JONES, H. R.: Pear leaf blister (*Taphrina bullata* Tul.). *Jour. Bath & West & South Co. Soc. Agr.* 18: 214–215. 1924.

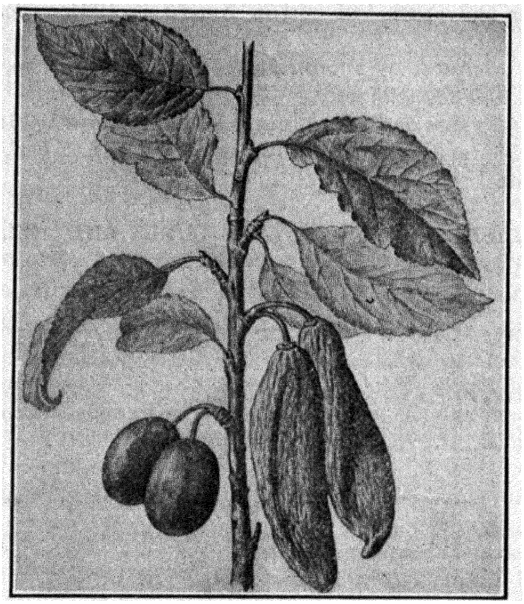

FIG. 147.—Plum branch showing two normal plums and two showing the effect of the plum-pocket fungus (*Taphrina pruni*). (*After Laubert.*)

Leaf blister or curl of oaks (*Taphrina cœrulescens* (M. & D.) Tul.).—WILCOX, E. M.: A leaf curl disease of oaks. *Ala. Agr. Exp. Sta. Bul.* **126**: 171–187. 1903. MARTIN, E. M.: Cultural and morphological studies of some species of Taphrina. *Phytopath.* **15**: 67–76. 1925.

CHAPTER XX

DISEASES DUE TO THE CUP FUNGI AND ALLIES

DISCOMYCETES

The fungi belonging to this group have no distinctive features of the mycelium, which is septate and generally within the substratum. Conidial fruits may be entirely absent or present and of various types, including aerial conidiophores, conidial tufts or acervuli. The characteristic feature of the group is the ascigerous fruit, or *ascocarp*, which shows great diversity of form but consists of an extended layer of *asci* or spore sacs, the *hymenium*, generally mingled with sterile hyphæ, or *paraphyses*, arranged in a palisade-like layer with supporting or accessory parts.

Types of Ascocarps.—Four general types of ascus fruits may be noted in the group:

1. A fleshy, waxy or gelatinous body, which usually is differentiated into a sterile stalk bearing an expanded ascigerous portion. This fertile part may be club shaped, short or long cylindric or irregular and the hymenial surface may be smooth, wrinkled or convoluted. In a few forms (Rhizina) the fruits are stalkless or sessile. This type is characteristic of the order *Helvellales*, most of which are saprophytic forms.

2. A fleshy, waxy or gelatinous body which is typically disk or saucer shaped or deepened to resemble a cup or vase, thus forming a typical *apothecium*. The hymenium occupies the upper face of the disk or the inner face only of the cup or vase forms. These apothecia vary from minute, microscopic forms to fruits 6 inches or more in diameter, and may be sessile or stalkless, or raised from the substratum by a sterile stalk. Fruits of this kind are characteristic of the order *Pezizales*, which includes the typical cup fungi.

3. A fleshy or leathery body which is globular or elongated, closed at first but opening later by a slit or by clefts which form star-like rays that turn back to expose the hymenium. The fruits may be free or sunken in a stroma and range from microscopic size to some 4 to 6 inches in length. Ascocarps of this type are characteristic of the order *Phacidiales*, which includes some important pathogenes.

4. A minute black body, leathery or carbonous, typically elongated boat shaped, which is free or becomes erumpent and with the maturity of the asci opens by a long, wide cleft to expose the hymenium. Ascigerous fruits of this kind are characteristic of the order *Hysteriales*, which furnishes a number of important pathogenes of coniferous trees.

In some of the simpler forms in which the development of the ascigerous fruit has been studied in detail, it originates following the union of nuclei of multinucleate oögonia and antheridia and is thus the result of a sexual process. The apogamous formation of ascigerous fruits, that is, without the intervention of a sexual process, occurs in many forms.

The asci are typically cylindric or club-shaped, eight-spored and in many species open by a lid or operculum which separates from the tip. The spores are forcibly expelled at maturity, simultaneous discharge of numerous asci giving rise to visible clouds of spores. The spores which are set free in this phenomenon of "puffing" are carried away by air currents, and thus reach new substrata on which they may germinate.

Classification.—The following is a tabulation of the orders and families with the most important genera furnishing plant pathogenes:

I. Helvellales

1. *Rhizinaceæ.* Ascocarp sessile.
   Rhizina. Ascocarp flat or crust-like with root-like outgrowths from the under surface; spores one-celled, hyaline.

II. Pezizales

1. *Helotiaceæ.* Disk of apothecium flat, saucer, cup or vase shaped. Peridium of hyaline, thin-walled cells. Spores variable.
   Sclerotinia. Apothecia originating from either free sclerotia or from mummied fruits of the host; spores one-celled, hyaline.
   Dasyscypha. Apothecia mostly sessile, disk bright colored, with delicate border or exciple, outside clothed with hyaline or colored hairs; spores hyaline, one-celled or finally becoming two-celled.
2. *Mollisiaceæ.* Apothecia free or becoming erumpent from the substratum, sessile, at first globose, becoming flattened. Peridium mostly of dark, thick-walled cells. Spores one to many-celled.
   Pseudopeziza. Leaf parasites forming bright-colored apothecia on leaf spots during the current season or on overwintering leaves; apothecia minute, breaking through the epidermis; spores one-celled, hyaline.
   Pyrenopeziza. On leaves and stems, forming dark apothecia, otherwise similar to Pseudopeziza.
   Fabraea. Similar to Pseudopeziza, but spores finally two or more, rarely four-celled, hyaline.
   Neofabræa. Similar to Pseudopeziza, but apothecia form in the old conidia-bearing stromata.
3. *Cenangiaceæ.* Apothecia immersed, later erumpent, dark, paraphyses forming an epithecium. Spores one to many-celled, hyaline or dark.
   Cenangium. Spores cylindrical to spindle form, often curved, one-celled, hyaline.

III. Phacidiales

1. *Phacidiaceæ.* Apothecia leathery or carbonous, depressed, globular or elongated. firm, dark or black, opening by rays or slits. Spores variable.
   Keithia. Apothecia separate, non-stromatic; spores ellipsoid or globoid, two to four-celled, brown.
   Coccomyces. Apothecia non-stromatic; spores filiform or needle-like, one to many-celled, hyaline.

Phacidiella. Apothecia flat, round; spores continuous, hyaline, elliptic; paraphyses forming a thick epithecium.

Trochila. Apothecia sunken and closed, later erumpent; spores continuous, hyaline, elliptic to oblong.

Rhabdocline. Apothecia elongated, subepidermal, non-stromatic, emerging by a longitudinal rupture of the epidermis; spores continuous, hyaline elongated ellipsoid, narrowed in the middle.

Rhytisma. Apothecia immersed in black stromata beneath the epidermis, elongated curved or round, opening by long slits; spores filamentous, or acicular, one-celled, fascicled and hyaline.

IV. Hysteriales

1. *Hypodermataceæ.* Apothecia black, elongated, opening by a longitudinal slit.

Lophodermium. Spores continuous, filiform and hyaline.

Hypoderma. Spores two-celled, cylindrical or fusiform and hyaline, ascus 8-spored.

Hypodermella. Spores continuous, elongate to spindle form, ascus 4-spored.

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BROWN ROT

Sclerotinia spp.

Brown rot is a serious disease of stone fruits and, especially in America, a trouble of minor consequence on the pome fruits. In its various phases in the typical development of the disease it attacks the blossoms, causing a *blossom blight;* the leaves and young twigs, causing a *leaf and twig blight;* the branches, with the production of *cankers;* and the fruit as it is approaching maturity or after it has been harvested, causing the rotted condition which has suggested the common name of brown rot. One or more phases of the disease may be in evidence during a single season and the behavior will vary more or less according to the environmental factors, the species or form and the hosts involved. The *fruit rot* has also been called mold, gray rot, soft rot, ripe rot and Monilia rot, and the affected fruits dry up and shrivel with the formation of "mummies," which hang on the tree or fall to the ground.

History.—*Sclerotinia fructigena* was first described by Persoon in 1796 under the name of *Torula fructigena* from fallen decayed fruits of plums, peaches and pears, while *S. cinerea* was not recognized until 1851, when it was first described as *Monilia cinerea* by Bonorden. The economic importance of brown rot was first recognized by von Thümen in 1875, who found it on half-ripe fruit on the trees. His observations were soon confirmed by Hallier (1876), and blossom and leaf blight of the cherry was reported in the United States by Arthur in 1886, by Galloway in 1889 and by Sorauer in Germany in 1891. The occurrence of jet-black mummies of apples was first observed by W. G. Smith in England in 1885. The study of E. F. Smith (1889) showed the importance of the blossom-blight phase of brown rot in peaches and also the penetration of the fungus into the wood with the development of cankers. Salmon (1910–1914) called attention to the blossom-blight and canker phases of the disease on the apple in England and later his studies were confirmed and extended by Wormald (1917). Jehle studied "peach canker" in New York and attributed it to *S. cinerea*, while McCubbin (1918), working in Ontario, decided that the brown-rot fungus was but the initial cause of cankers which were extended by the inroads of another fungus, *Valsa leucostoma.* Following the initial observations on the various phases of the disease, there were numerous contributions by workers in the various countries.

In 1893 Schroeter recognized the ascomycetous nature of the two species of fungi causing brown rot, and placed them in the genus Sclerotinia. Woronin (1898) showed conclusively that *S. fructigena* and *S. cinerea* were distinct species, but for many years many other investigators did not accept his conclusions and referred the brown-rot fungus to the single species, *S. fructigena.* Again in 1908 Aderhold called attention to the differences in the two species, and in 1913 Matheny showed that the American brown rot is *S. cinerea,* and his conclusions were confirmed by other investigators. The researches of Wormald, begun in 1917, have not only shown that *S. fructigena* and *S. cinerea* are distinct, but have demonstrated the existence of two forms of *S.*

cinerea, forma *mali* and forma *pruni*, and recognizable differences between the European and American strains of *S. cinerea*. Wormald's forma *americana* has been raised to species rank by Norton and Ezekiel (1924).

A form on apricots was named *Oidium laxum* by Ehrenberg in 1818, but this was transferred to Sclerotinia by Aderhold and Ruhland in 1905. Wormald has been unable to separate this morphologically from *S. cinerea* (1921). *Monilia oregonensis* Barss and Posey (1923) is *S. cinerea* forma *pruni*.

Apothecia of brown rot were first discovered by Norton in the peach and plum orchards of Maryland in 1902. This stage was reported by Aderhold and Ruhland in Europe in 1905 from mummied apples and apricots, and descriptions were given of the apothecial forms of *S. fructigena*, *S. cinerea* and *S. laxa*. Apothecia were first found in England in 1920 by Wormald, on apricots in New South Wales by Harrison in 1922 and in New Zealand by Cunningham in 1922. The more recent studies seem to indicate that *S. fructigena* of England and the Continent does not occur in America. These discoveries of the apothecial stages showed the validity of Schroeter's references of the Monilia forms of the brown-rot fungi to the genus Sclerotinia. A most complete historical presentation of the brown-rot problem will be found in the series of articles by Wormald (1917–1921), recent contributions by Roberts and Dunegan (1924) and Ezekiel (1924).

Geographic Distribution.—Brown rot in some form is known in practically all countries of the world wherever stone fruits are grown, but its occurrence is limited by climatic factors. It has been epiphytotic in some seasons in Europe, America, Japan, Manchuria, Australia and New Zealand and is reported in its conidial forms from South Africa. The disease has been especially severe in the peach-growing districts of the Atlantic Coast states from the New Jersey southward. Delaware, Maryland, Alabama and Georgia have suffered heavy losses, but it has probably been most destructive in the last-mentioned state. In the drier regions to the Southwest it has been absent or rare. It occurs regularly in the more northern states east of the Rocky Mountains and is serious as far north as Ontario on stone fruits and occurs to some extent on apples. The disease is rare in California except in the region of San Francisco Bay and a few other moist localities. It is rare or unknown in the dry sections of the Pacific Northwest east of the Cascades, but is serious on cherries, prunes and peaches in the humid sections of western Oregon and Washington. It has never been reported from the important irrigated districts of central Washington. In California the apricot is also affected by a green rot and twig blight due to *S. sclerotiorum* (*Lib.*) Mass., which has no conidial stage. The severe development of brown rot may be expected in any warm, humid section and is most severe in seasons of abundant insect infestations. Fruit rot is promoted by the curculio injuries and Wormald has shown that the wither-tip disease of plums in England in 1916 and 1918 was associated with the attacks of aphids.

Symptoms and Effects.—Under certain conditions the direct attacks of the blossoms is a very serious phase of the disease. The flower parts turn brown prematurely and during moist weather appear soft and rotting, but under dry conditions are only withered and discolored. The pistil may be blighted first or the discoloration may start in the petals and advance down the claws into the calyx cup. In these parts the first evidence of the progress of the disease may be the appearance of brown or sometimes nearly black discolored areas occupying one side. As the disease progresses the entire calyx cup blackens and the discoloration may extend through all the flower parts and down into the pedicels. An

examination of blighting blossoms during humid periods will frequently show whitish or tawny fungous tufts over the surface of the affected parts, and a similar development of the parasite may be induced by placing the blighted flowers in a moist chamber for 24 to 48 hours. *Blossom blight* is next in importance to the fruit rot as a destructive phase of this disease. There may be only a slight touch of the trouble in an orchard, or it may be so severe as completely to ruin the prospects of a crop. Light attacks of blossom blight are frequently overlooked, and some of the more severe outbreaks are sometimes attributed to other agencies.

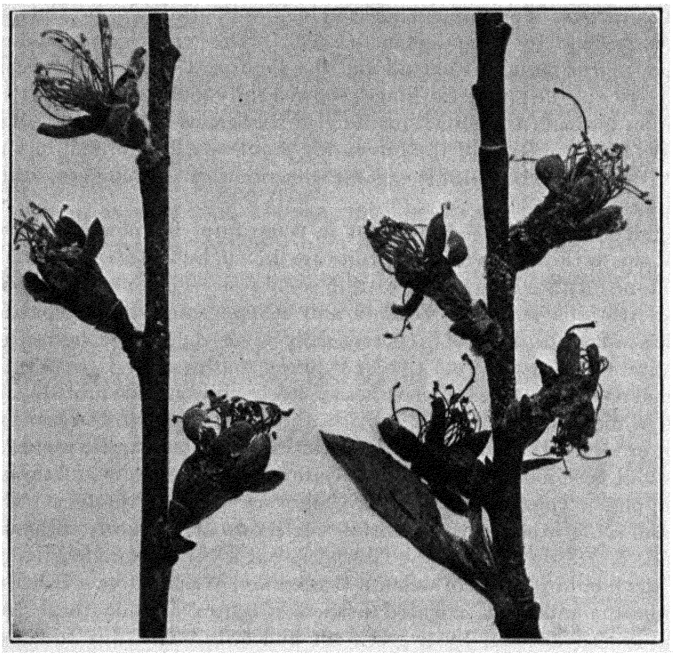

FIG. 148.—Blossoms of peach blighted by the brown-rot fungus. (*Photograph by E. E. Honey.*)

Leaf and twig blight (*wither tip* of English writers) are not so common as the blossom blight. In the latter the infection may advance into the fruit spurs and cause blighting of adjacent structures, but probably the majority of flower infections cease to advance after the base of the flower pedicel has been reached. Direct attacks of young leaves or of succulent shoots may occur just following the blossoming period. In leaf attacks the affected areas turn brown, no circumscribed spotting results, but the discoloration spreads from the point of infection until the whole leaf or an extended area is involved. Such affected leaves and shoots shrivel as they dry up and remain hanging as blighted or blasted structures, with an appearance similar to that of frost injury. In wet seasons this phase of the disease may be so severe on some varieties as to give the trees the appearance of being infested with fire blight.

From blighted twigs or fruit spurs the infections may spread into the larger limbs, causing dead areas of bark, which later become more or less open wounds or *cankers*. This phase of the disease is especially common on the peach in certain localities. On this host young cankers first show a sinking and discoloration of the tissue beneath the bark, which is later followed by cracking and splitting and more or less exudation of gum. Callus is formed around the diseased area and the wound may be healed or the callus may be invaded and the lesion increased in extent. Such cankers may continue to extend for a period of years before the branch is girdled or the progress of the disease checked. In cankers that are not of too long standing, the remains of a fruit spur through which the pathogene entered may be found occupying a central position. Brown-rot

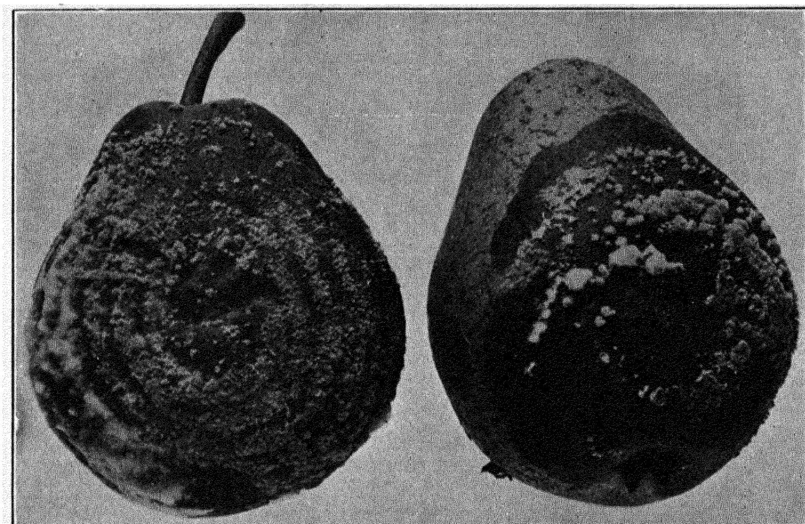

FIG. 149.—**Pears rotted by the common American brown-rot fungus, with concentric rings of conidial tufts.** (*Photograph by E. E. Honey.*)

cankers are more frequent in Europe than in America. In the former they are common on cherries, plums and apples, while in America they have been frequently observed on the peach in the eastern United States and to a less extent on prunes and pears in the Pacific Northwest.

The *rotting of the fruit* is the most serious and destructive phase of this disease. Young fruits may be attacked at any period following blossoming, but if they escape blossom blight, extensive infections are not likely to occur until the fruit is approaching maturity. Under dry conditions but little fruit rot will be noticeable in the orchard, but the disease may cause much rotting in fruit that is in transit to market or is being held for consumption. The first indication of the disease on the fruit is the appearance of a small, circular brown spot, which rapidly advances if warm, moist conditions prevail. The rapidity of advance will be influ-

enced by the age of the affected fruit, being more rapid the nearer the fruit is to maturity, but the lesion extends until finally the whole of the fleshy portion is involved. In the stone fruits there may be more or less exudation of gum, and insect injuries (curculio) through which the infection occurred may also be evident. The rotting spots do not become sunken and there is generally no shriveling until the entire fruit is rotted. A gradual shriveling and wrinkling follows and the affected fruit is transformed from the soft, watery, rotten condition into a dry, shriveled mass, a so-called "mummy," which resists further decay.

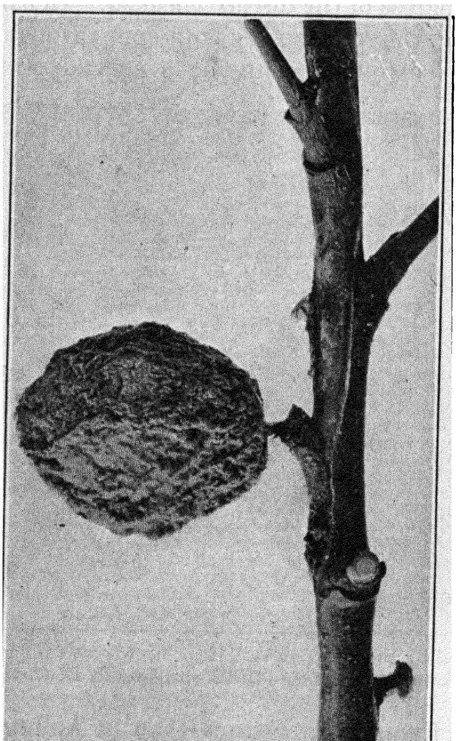

FIG. 150.—Peach mummy and branch, showing a young canker caused by the growth of the fungus down the fruit pedicel. (*Photograph by E. E. Honey.*)

Young lesions show a smooth unbroken epidermis, but later, when the lesion has extended, whitish, gray or pale-brown tufts may burst through the skin. The extent of this superficial development will vary according to moisture conditions, and the species or strain, from none under extremely dry conditions to a complete covering with the gray or tawny velvety tufts under conditions of abundant moisture. The decayed fruits may drop from the tree, but many remain hanging and in stone fruits are frequently cemented into clusters, which are evident

during the following fall and winter. Under average conditions the majority of the affected fruits fall to the ground, but these are also transformed into mummies, which lie on the surface or become covered with soil or litter. A core rot of early varieties of apples has been described (Dowson, 1926) due to *S. fructigena.* In some cases there is no external evidence of the rot, while in others there may be a small brown spot at either calyx or stem end. A marked deviation from the typical behavior as described above is to be found in certain apple infections. The fruit may become a jet black, and the skin assume a shiny aspect, with little wrinkling or reduction in size and no external evidence of the pathogene. This "Schwarzfäule" of the apple may appear in the orchard, but it has

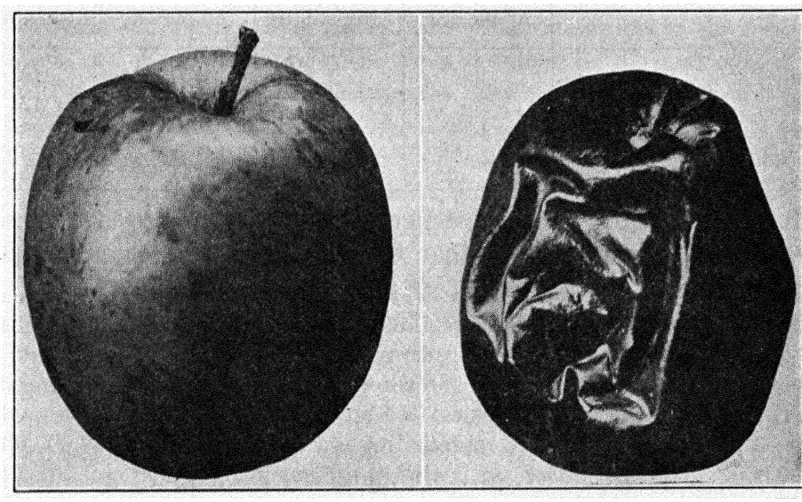

FIG. 151.—Normal apple and coal-black mummy produced by brown-rot fungus. This is a storage form of brown rot.

been found most frequently in storage. It can be distinguished from the true black rot (*Physalospora malorum*) by the following:

Brown rot: Fruits coal black, smooth shiny, little wrinkled or reduced in size.

Black rot: Fruits become black from numerous minute black pustules beneath the skin, shrivel rapidly and become much wrinkled and reduced in size. Either type of rot may be brown at first and assume the black color later.

Losses from Brown Rot.—The injury from brown rot is due to the following: (1) the blighting of blossoms, with consequent failure of affected trees to set fruit; (2) the blighting of leaves and twigs, with the loss of these as a part of the normal tree; (3) the formation of the cankers which may interfere with the life of a branch or girdle it and cause the death of all distal parts; and (4) the rotting of the fruit in the orchard or after harvest. Under extreme conditions blossom blight may cause

almost a complete failure, while a loss of one-third to two-thirds of the blossoms has been frequently reported for stone fruits. Unsprayed orchards of stone fruits are sometimes almost a total loss due to the rotting of the fruit in the field, while shipments which appear sound at packing time may reach the market in a worthless condition. The extent to which losses may fall upon the transportation companies, commission men and consumers after the fruit leaves the producer is emphasized by the following figures (Brooks and Fisher, 1921):

PER CENT OF ROT

	Sprayed		Unsprayed	
	At picking	After storage	At picking	After storage
Prunes	1	2.5	4.25	16
Prunes	1	2.0	6.00	13
Prunes	1 5	7	4.75	28
Cherries	4	..	16 00	

It may be noted from these figures that the unsprayed fruit went down in storage more rapidly than the fruit from the sprayed orchards.

Brown rot probably causes more loss to peach growers than all other maladies of the peach combined, with perhaps the exception of "yellows," which kills the tree outright. In the South the brown rot often causes the destruction of half or even practically all of the crop, and throughout the territory under consideration (United States east of Rockies) the annual shrinkage in yield is perhaps 25 to 35 per cent of the crop, representing a valuation of about $3,000,000 to $4,000,000. Although brown rot is always present in the peach orchards of the humid sections, causing a rotting of a certain proportion of the fruit, it becomes notably destructive only under certain weather conditions, when within a period of 10 days or 2 weeks it will spread so rapidly as to result in the destruction of practically the entire crop. Such disastrous outbreaks are likely to occur during moist, humid weather as the fruit begins to ripen. The brilliant prospects of the orchardists are thus within a few days obliterated as if by fire (Scott and Quaintance, 1911).

Etiology.—Brown rot is caused by species of Sclerotinia which produce their conidial or *Monilia* stages on the various parts of the susceptible hosts and an apothecial stage on the overwintered mummies. The most complete studies of the relationship of the different fruit Sclerotinias has been furnished by Wormald. According to this author and some more recent reports the following gives the present status of our knowledge.

Sclerotinia fructigena (Pers.) Schr. Causes fruit rot of apples, pears, plums and cherries and on apple trees may produce cankers. Present in England and on the Continent. Not known in America.

S. cinerea (Bon.) Schr. On apples, pears, plums, peaches, cherries and probably on apricots.

Forma *mali*. Produces blossom wilt and canker disease of apples. This strain is confined to Europe.

Forma *pruni*. Produces fruit rot, blossom wilt or blight, wither tip or twig blight of plums and cherries. This strain has been definitely recognized as occurring in Great Britain; also on the Continent and in several American localities.

Forma *laxa*. On apricots only. Some doubt exists as to the apricot strain being distinct from *pruni*.

S. americana (Worm.) N. and E. Produces the characteristic effects on stone and pome fruits throughout America and in Australia and New Zealand. This has recently been raised to the rank of a species by Norton and Ezekiel (1924) who propose the name *S. americana* (Worm.). Roberts and Dunegan (1924, 1927) have called attention to the fact that the name of our common brown-rot fungus should be *S. fructicola* (Wint.) Rehm if it is to be raised to specific rank, since this name was first given to American collections, a feature which was pointed out by Pollock (1909). Wormald (1928) objects to *S. fructicola* for *S. americana* because there is no conclusive evidence that Winter's description referred to *americana*.

A form causing typical brown-rot effects on stone fruits and pears on the Pacific Coast described as a new species by Barss and Posey (*Monilia oregonensis*) has been shown by Ezekiel (1924) and Wormald (1928) to be the same as *S. cinerea* forma *pruni*.

Wormald does not recognize the cherry brown rot described by Westerdijk as distinct from *S. cinerea*. *Monilia cinerea cerasi* of sour cherries and *Monilia cinerea avium* of sweet cherries described by Killian (1921) are to be separated with difficulty from the forms described by Wormald.

The tufts of the fungus which appear on the diseased parts under moist conditions are made up of groups of conidiophores, sometimes rather loosely aggregated but more generally formed into fairly compact pustules. The formation of the conidial tufts does not begin until the substratum is well permeated by the mycelium and then may be sparse or lacking if the air is very dry. The conidial stages of *S. fructigena* and *S. cinerea* may be recognized by the following characters (Wormald):

S. fructigena. Conidial pustules buff yellow, conidia average 21 by 13μ, barren in winter or producing non-viable conidia; conidia germinate on prune juice to produce a long germ tube, 600 to 1000μ, before branching.

S. cinerea. Conidial pustules gray, smaller than in *fructigena*, general average winter conidia 11.5 to 8μ, summer conidia 17 to 11μ; conidia germinate on prune juice to produce germ tube with branches while still short.

Mathaney gives the following measurements and form of the conidia:
S. fructigena, 22.1 by 11.2μ; 665 measured; form elongated ellipsoid.
S. cinerea, 14.4 by 10.8μ; 181 measured; form more rounded.
S. cinerea americana, 14.7 by 11.9μ; 942 measured.

The conidiophores arise as short hyphæ which soon become septate at the extremities, branched and nodulose. The branching proceeds in an indefinite and usually irregular or semidichotomous fashion. From the apex of these branches towards the base, conidia are rapidly cut off, these cells remaining for a time simply moniliform or as branched chains, each constriction between the nodulations eventually marking the line of separation between adjacent spores (Duggar).

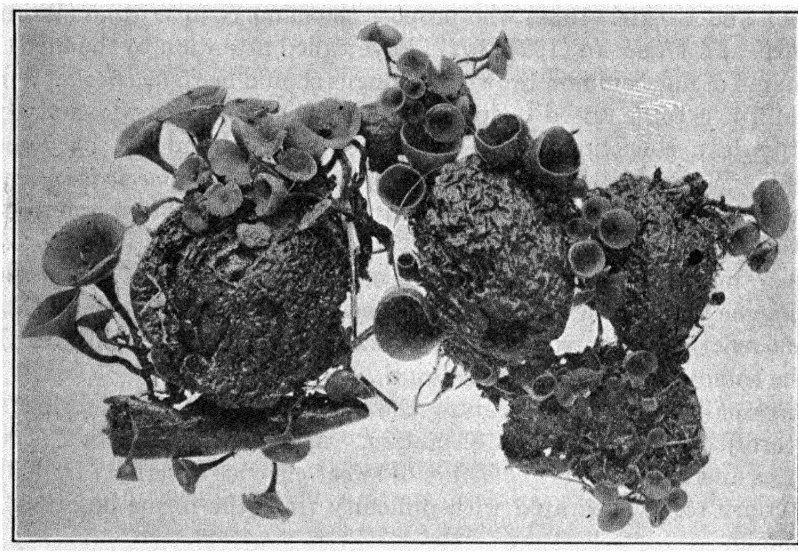

FIG. 152.—Apothecia of the American brown rot developed in the spring from peach mummies. (*Photograph by E. E. Honey.*)

Microconidia ranging in size from 2.55 to 3.22μ and spherical in form were described by Woronin for both European forms, and these have been observed by Valleau (1915) for the American form in cultures and on plums.

Dense aggregates of mycelium or sclerotia which are formed in the mummies give rise to the apothecial fruits which reach maturity under normal conditions just about the time the host is in bloom. The mummies may be on the surface of the ground and more or less covered by litter or buried at various depths in the soil. Each mummy may produce from one to 20 or even more (Norton *et al.*, 1923) brown cup-like disks 2 to 15 millimeters (5 to 8 millimeters general) in diameter, each raised on a cylindrical stalk or stipe, which is dark brown below and lighter above. The stipe is more or less sinuous and varies in length with the depth of the mummy below the surface of the soil. The average length is 0.5 to 3

centimeters but the formation of a single apothecium has been recorded from mummies 2 and 3 inches deep. The disk, which is at first campanulate, widens out until cup-shaped or even flat, and in old specimens may be torn and with recurved edges. The inner face of the disk is occupied by the hymenium or a layer of erect asci and paraphyses. The asci are eight-spored, cylindrical-clavate, with an apical pore, and are mingled with hyaline, septate, simple or branched paraphyses with slightly swollen tips. The ascospores are hyaline, and uniseriately or subbiseriately arranged in the upper half of the ascus. The measurements given for the American form by different workers vary from 89 to 190 by 6 to 10.8μ for the asci and 4.1 to 16.7 by 3.1 to 8.1μ for the ascospores.

While the asci and ascospores of the European *S. fructigena* and the American form apparently correspond in size, there are differences which remain distinct. The ascospores of the former are sharply pointed at each end and are free from oil droplets, while the ascospores of the latter are rounded at the ends and possess oil droplets (Matheny, 1913).

It was at first believed that the mummies did not produce apothecia until they had passed the second winter in or on the ground, but Roberts (April, 1921) and Ezekiel (December, 1921) demonstrated that "under natural conditions apothecia may develop the spring following inoculation. Cold is probably a factor influencing the production of apothecia, as apothecia were produced in 25 weeks from chilled mummies." Previously, Reade (1908) had reported that the brown-rot fungus "has been grown on artificial media from ascospores to ascospores again, and completes its life cycle in 1 year." Norton *et al.* (1923) report the heaviest production of apothecia from mummies on or close to the surface of the ground, while none were formed on mummies covered with 2 inches of soil. Their experiments have shown:

(1) That burying brown-rot mummies below the surface of the soil prevents the production of apothecia, the precise depth necessary doubtless varying with the type of soil; and (2) if such buried mummies are brought again to the surface of the soil, they are still able to produce apothecia the next spring.

Pollock observed the production of apothecia from 10-year-old plum mummies. This behavior of mummies has an important bearing on control, since it must be obvious that mummies unless buried deeply in plowing will still be able to produce apothecia if the ground is replowed and they are brought up to the surface. The opinion seems to prevail that mummies 2 years old or more are more likely to produce apothecia than at the end of 1 year, the conditions necessary being sufficient moisture, favorable temperatures and proper aeration. More recently Ezekiel (1927) reports that peach mummies (*S. americana*) buried 2 to 8 inches under orchard conditions disintegrate in less than 10 months

sufficiently to prevent apothecial formation but that plum mummies are more durable.

When the apothecia reach maturity the ascospores are forcibly expelled. This phenomenon of "spore puffing" can be readily observed if fresh fruits are held in a moist chamber for a time and then exposed to drying or drafts of air. A visible cloud of spores may be seen to rise from the surface of the hymenium due to the simultaneous explosion of numerous asci. This is the normal method by which spores are expelled under natural conditions and they are carried away by convection currents and the wind. It is an effective adaptation that brings the ascospores to maturity and expels them just at a time when the flowers of the susceptible hosts are ready for infection.

The source of the spores which produce the first spring infections may be (1) conidial tufts: (*a*) from fallen or hanging mummies; (*b*) from cankers or blighted twigs; and (2) apothecia from the mummies of the last or previous seasons. The first spring infections may be blossom blight or wilt or leaf and twig blight. It has been shown that the conidia of the common American brown rot retain their vitality during the winter even in regions as far north as Illinois (Conel, 1914) and Vermont (Bartram, 1916), and a similar behavior holds for the European *S. cinerea*, but it has been shown that the conidia of *S. fructigena* do not survive winter conditions.

When the fruit is half grown or over, the following are the possible sources of the conidia which may start the fruit rot: (1) mummied fruits of the previous season; (2) blossoms blighted in the spring of the current year; (3) blighted leaves killed earlier in the season; (4) blighted twigs killed during the previous season; (5) cankers formed previous to the current season. From one or several of these sources conidia are carried by the winds and rains to the developing fruits. These conidia germinate at once and the germ tubes enter the fruit, their penetration being facilitated by other fungous lesions (scab) and by insect injuries, especially by curculio. Under warm, moist conditions the mycelium develops with great rapidity and an infected fruit is soon completely rotted. The first conidial tufts soon appear and more spores are formed, which will be carried away to other fruits to continue the work of destruction. The mycelium may grow from one infected fruit into an adjacent fruit with which the first is in contact, and thus a whole cluster of fruits (stone fruits especially) may be cemented into a mummified group. At harvest time spores may be generally prevalent in the orchard and be lodged on the surface of the fruit, or incipient infections may already have taken place, and development continue during transit to market or before consumption. It is undoubtedly true that bruises made in handling such soft fruits as peaches frequently furnish the avenue for the entrance of the pathogene which is just lying in wait for the opportunity.

Predisposing Factors.—The extreme variation in the severity of brown rot in different regions and in the same region during different seasons emphasizes the influence of environmental factors. Rainy periods with relatively low temperatures at the time of blooming are conducive to blossom blight, which is sometimes mistaken by orchardists for frost injury.

Moisture not only favors the growth of the fungus and the production and germination of the spores, but it also renders the fruit tender and watery, and therefore more susceptible to rot. In a dry season, or in one with only occasional rains of short duration, a peach crop may be expected to reach maturity practically free from rot, particularly if the weather is cool; but when a series of cloudy days with frequent showers occurs about picking time, half or even all of the crop may be destroyed by brown rot. Prolonged cloudy, drizzly weather, even though the precipitation may not be great, is far more dangerous than a heavy rain followed by clearing. Hot weather also favors the rapid growth of the fungus and increases the danger of its destroying the crop (Scott and Ayres, 1910).

The transportation losses are affected very much by the temperatures to which the fruit is exposed and therefore by the length of time which elapses between packing and refrigeration. The brown-rot fungus makes a relatively slow growth at temperatures of 10°C. or lower, but a rapid growth at higher temperatures until the optimum is reached.

It is evident that in unfavorable weather success with long-distance shipments requires not only a low car temperature upon arrival at destination but a low temperature from the time the peaches are packed and as much coolness as possible from the time they are picked (Brooks and Cooley, 1921).

The brown-rot fungus appears to be able to enter the fruit through the unbroken skin via stomata or lenticels and, more rarely, through the epidermis (Curtis, 1928), but infections are facilitated by breaks or bruises. In the peach districts which suffer such heavy losses from brown rot, peach scab is generally common and the curculio is abundant. Cracks made by this fungus or feeding or oviposition punctures made by the curculio are very common avenues of entrance. Various insects feeding on susceptible structures may not only make the wounds but possibly may be responsible for inserting the spores, although these are generally prevalent in brown-rot regions and would be lodged in open wounds by other means. The curculio is undoubtedly the worst offender, especially in sprayed fruit, since in certain tests it was found to be responsible for 93 per cent of the infections (Scott and Ayres, 1910).

Host Relations.—Brown rot affects all stone fruits, peaches, plums, prunes, sour and sweet cherries, apricots and nectarines; also the pome fruits, apples, pears and quinces. In general, it is much more serious on the stone fruits than on the pome fruits, and in America may be considered one of the minor apple diseases. The European brown rot (*S.*

fructigena) is one of the most serious apple rots in England. Recently *S. fructigena* has been described as causing rotting of 50 per cent of medlars in Italy and has also been reported as the cause of rot of grapes in Czechoslovakia. It is suggested that this form on grapes may represent a new biologic form.

Our knowledge concerning the susceptibility of varieties is still rather meager. Summer varieties of apples, like the Yellow Transparent and Chenango, are reported as very susceptible. Genet is reported as severely attacked in Kentucky. Cherries appear to suffer less damage than peaches or plums, and sweet cherries are generally reported as more susceptible than sour cherries. Governor Wood and Heiderman sand-cherry are listed as susceptible, and the Montmorency as relatively resistant, although less so than native varieties.

It would appear that among peaches the sorts densely covered with hairs or down, such as Alexander, Hills' Chile and Triumph, are unusually susceptible. Among the more resistant sorts are to be found the Carmen, Early Crawford. Elberta, Chinese Cling and some others (Duggar, 1909).

The observations of McClintock (1921) on the prevalence of blossom blight of peaches is of special interest:

Since the early varieties always blossom several days later than the late maturing varieties they are more liable to blossom blight because of additional time for the fungus to develop conidia with which to infect the blossoms.

Blossom blight was serious only on early varieties, including Mayflower, Red Bird, Early Rose, Alexander, Victor and Early Carmen. Little or no blossom blight was observed on Carmen, Hiley, Georgia Belle, Elberta or Hale.

Varietal resistance of plums has been studied by Valleau (1915) and he concludes that:

Resistance is apparently correlated with (*a*) a thick skin; (*b*) the production of parenchymatous plugs which fill the stomatal cavity; (*c*) the production of corky walls in the lining cells of the stomatal cavity; and (*d*) firmness of fruit after ripening. There seems to be no relationship between oxidase content of the fruit and resistance or between tannin content and resistance.

The most resistant of the varieties tested were an Abundance × Wolf 18, Burbank × Wolf 9, Burbank × Wolf 15, South Dakota Nos. 2 and 3, which were probably sand-cherry hybrids, Reagan (Wayland × *P. americana*) and American Seedling No. 1. Burbank was more susceptible than Wolf, but not so susceptible as some other varieties. Abundance and Satsuma have been reported as susceptible, while Hansom, Clinton, Forest Rose, Indiana, Miner and Prairie Flower are listed as resistant varieties. Since resistance appears to be correlated with firmness, and susceptibility with a soft and tender texture, the best varieties are susceptible while the resistant varieties are of minor importance.

The morphological aspects of resistance have been studied more recently by Curtis (1928). In the case of plums having a weak cuticle and external epidermal walls, the morphology and number of stomata are of no great importance; but if the cuticle is resistant, then the total number of stomata is of more significance in measuring resistance than their structure. Penetration may take place even in the absence of either stomata or lenticels, as in the Yellow-Cherry plum. The hypoderm also plays a part in resistance, but contrary to Valleau the formation of parenchymatous plugs and stomatal lenticels is considered unimportant. According to Willamen (1926), sufficient evidence is now available to warrant the use of the skin test alone in the determination of resistance of plums to brown rot.

Susceptibility to brown rot is known to increase with maturity of the fruit. Softening during ripening is due to the solution of the middle lamellæ, and "the absence of the middle lamella in fruits which have softened owing to ripening explains the greatly increased spread of the disease at ripening time" (Valleau), since the hyphæ advance entirely in the intercellular spaces. The idea has been prevalent that young fruits are more resistant because of greater acidity, but Hawkins (1915) has shown that the acid content of rotted peaches is greater than that of normal ones.

Control.—The brown rot in America is rarely of sufficient severity on pome fruits to justify special control measures, but in many environments the stone fruits must be protected in order to insure a crop. The actual control practices must be suited to the environment and the phases of the disease that are generally prevalent. In some environments the fruit rot will be the only phase of the disease, while in others blossom blight or other phases will be of frequent if not constant occurrence. No single practice is adequate and the available control measures may be listed under the following heads:

1. *Sanitary Measures.*—These are practices designed to remove the sources of spore production and so reduce the prevalence of spores in the orchard. In the ordinary pruning operations care should be taken to *remove all blighted twigs.* If cankers are present they should be given attention, either by removing the branch which bears them, if it can be sacrificed without serious detriment to the tree, or by treatment, if on large limbs. In the latter case they should be cleaned out by the removal of all the diseased wood and bark a short distance beyond any evidence of diseased or discolored tissue, and after drying of the wound a coating of coal or gas tar should be applied as a waterproofing and a disinfectant (Jehle, 1914). The removal of short fruit spurs on large limbs is also advisable, as infections on these are very frequently responsible for the initiation of cankers. Trees which have a dense head should be pruned by thinning out rather than heading back, since an open growth gives a

better circulation of the air and penetration of sunlight, which will facilitate rapid drying of moisture and lessen the chances of infections. Thinning of fruit clusters is also of value, and all mummied fruit that appears on the trees should be removed and destroyed. In some localities

Growers have made it a practice to patrol the orchard during the summer, examining each tree and knocking out all brown-rot clusters with a long pole provided at the top with a hook. This decayed fruit and any that may have dropped of its own accord is collected and destroyed (Barss, 1923).

It would seem that this would be a better practice than allowing the clusters of mummies to remain until regular winter pruning. Some orchardists make a practice of raking up and removing all fallen fruit from the orchard either just ahead of the pickers or after the harvesting has been completed. This is of value for brown rot alone, and is doubly valuable in those regions where the curculio is an important factor, but in such cases should be begun early when dropped fruit is still small.

The best way to destroy these fallen peaches is by burying deeply with quicklime. They should be placed at least 24 inches below the surface. Growers have found a long trench that can be extended from time to time useful for the destruction of the "drops." The "drops" should be covered with soil after the quick lime has been added (*U. S. Dept. Agr. Circ.* **216**).

In case of brown rot alone, the lime is not necessary and fruit buried 3 inches below the surface will never be a source of danger.

2. *Cultural Practices.*—Plowing and harrowing are of great value in the control of brown rot. Fruit that is deeply buried is not likely to produce apothecia. Harrowing in the early spring and during the blossoming season is likely to disturb the partly formed apothecia and prevent their development. Orchards that are not plowed or cultivated until after the blossoming season, as is frequently the practice, usually have an abundance of apothecia, while those that are plowed or cultivated before that time are usually practically free from them, except in the tree rows or other unplowed areas. Orchards free from apothecia, made so through cultivation or otherwise, have been found to be free from blossom infection and usually relatively free from brown rot (Fisher and Brooks, 1924).

The above statement applies to the prune orchards of the Pacific Northwest, where the apothecia seem to be a most important source of the early infections, and it probably holds for other sections also (Ezekiel, 1926). When plowing and cultivating are consistent with the horticultural demands of an environment, they will undoubtedly be of value.

3. *Spraying or Dusting.*—In regions of severe infestation, spraying or dusting must be a regular practice as an insurance, but the number of applications of fungicide and the formula used should be varied to suit local conditions. The following applications have been recommended:

a. Before the blossom buds burst or come into bloom (the bud spray); fungicide alone.

b. When most of the petals have fallen (calyx spray); fungicide plus insecticide for curculio if present.

c. When calyces or "shucks" are shedding or as soon as shed; fungicide plus insecticide (shuck spray).

d. About 2 weeks after the shuck spray; fungicide plus insecticide.

e. The last application has been varied from 1 month to 2 or 3 weeks before ripening (fruit spray); fungicide plus insecticide. In some regions when the fruit spray is given 1 month before ripening, additional protection of the market product is afforded by a dusting with sulphur 10 days before harvest.

When blossom and leaf and twig blight are not of consequence, the spraying may be begun with application *b* and the fungicide used only in the last two applications. The recommendation for prunes and cherries in the Pacific Northwest omits application *d* and the insecticide, since curculio is not present.

Control of brown rot is inseparably connected with curculio control when that insect is present. One pound of powdered arsenate of lead to each 50 gallons of water is to be used whenever an insecticide is indicated or 5 per cent of arsenate of lead if the dust formula is employed.

The following liquid fungicides have been recommended: (*a*) Bordeaux, 4–4–50, or sometimes 3–4–50, 2–3–50 or 1½–2½–50; (*b*) lime sulphur, 1 to 50; (*c*) self-boiled lime sulphur, 8–8–50 or some other sulphur fungicide. Since its first introduction (Farley, 1923) dry-mix sulphur lime has been very commonly used, especially for peaches, since it is much easier to prepare than self-boiled lime sulphur and also can be used without injury to fruit or foliage. For prunes (*a*), (*b*) or (*c*) may be used; for cherries, (*a*) or (*b*), but not self-boiled lime sulphur on sweet cherries because of its dwarfing effect; for peach and Japanese plums use only (*c*); for apricots (*a*), the weaker strength, or (*b*). Under California conditions self-boiled lime sulphur, if used after the fruit had set, caused pronounced dwarfing of apricots.

The results with dusting have been somewhat variable, but dusting has been used mostly for peaches. In some cases dusting has given better fruit than spraying. The most generally recommended formula is 80 per cent dusting sulphur, 5 per cent arsenate of lead and 15 per cent hydrated lime.

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ANTHRACNOSE OF CURRANTS

Pseudopeziza ribis Kleb.

This is a disease of currants which causes a spotting of the leaves and shows to a lesser extent on petioles, young canes, fruits and fruit pedicels,

thus causing injury both directly and indirectly to the foliage and fruit. It is also called the leaf blight and leaf spot, but should not be confused with the leaf spot of currants due to an entirely different fungus, *Mycosphærella grossulariæ.* The two can be readily distinguished by superficial characters, the lesions of Pseudopeziza leaf spot being small, while those of the Mycosphærella leaf spot are usually ⅛ inch or more in diameter.

History and Geographic Distribution.—Anthracnose was first noted by Dudley as a serious disease of currants in America in 1889, although it had been reported somewhat earlier. It was first reported in America from Connecticut by Berkeley in 1873 on black currants, and later by Peck on the leaves of the fœtid currant (*Ribis prostratum*) in the Adirondack Mountains. Following the serious development of the disease in New York in 1889, it did not again become epiphytotic until 1901, when it was studied by Stewart and Eustace. It was not found by Pammel in Iowa in 1895 when he was making a special study of the Mycosphærella leaf spot of currants, but appeared there somewhat later. It was noted in Oregon in severe form at least as early as 1911–1912, and was epiphytotic in Washington in 1915, but has not been very serious in Washington since that time. The disease was known in Europe as early as 1867, and it has been reported also from Asia and Australia, but the country in which it originated is uncertain. While the anthracnose is rather widely distributed in the countries mentioned, it is of more local occurrence than many other fruit diseases and rather variable in the severity of its attacks. Noteworthy contributions to the life history of the causal fungus and on the control of the disease were made in Germany by Klebahn in 1906 and by Ewert in 1907. Stewart contributed valuable information on the control of anthracnose in nursery stock by use of Bordeaux or lime sulphur in 1915 and by dusting with sulphur in 1916.

Symptoms and Effects.—The disease first appears on the older and lower leaves of a bush causing minute, dark brown, circular or subcircular spots, about $\frac{1}{25}$ inch in diameter, and most conspicuous on the upper surface. The lesions may be few in number or so numerous that they may coalesce and cause larger and more irregular dead areas involving much of the leaf blade. Shiny, translucent, whitish or flesh-colored masses may be noted on the surface of the spots, appearing as minute specks at their center, but these spore masses may be washed away by rains. When the leaf spots are few in number, the balance of the leaf may remain apparently normal, but when they are numerous there may be a pronounced chlorosis of the intervening leaf tissue, and the affected bushes will show a decided yellow color, frequently recognizable for a considerable distance. During some seasons the disease may confine its attacks to the lower foliage, but under the most favorable conditions the infections become progressively more abundant until the upper and younger foliage is also involved. In light attacks only a few lower leaves are dropped, but in the severe infections which cause pronounced chlorosis there is a progressive defoliation from below upwards, until almost complete defoliation may result. This condition may be reached by midsummer, and in recorded epiphytotics the canes were bare with the exception of small tufts of leaves at their tips.

While the infections are most evident upon the leaf blades, lesions may also occur on the petioles and upon the one-year-old canes. The lesions on the leaf stalks are evident as conspicuous, black, slightly sunken spots, and contribute to the injuries inducing leaf fall. The lesions on the canes are light brown or pale yellow and rather inconspicuous except when numerous, and apparently cause little or no injury, but are of especial significance in the life history of the parasite.

In severe attacks the fruits and fruit stalks are also directly affected. On the berries the lesions are evident as minute circular black spots resembling fly specks. The fruit lesions are noticeable on the green berries, but become much less conspicuous on the ripening berries. In severe fruit infections some of the most severely affected berries may split or crack and considerable shedding may result before ripening is completed.

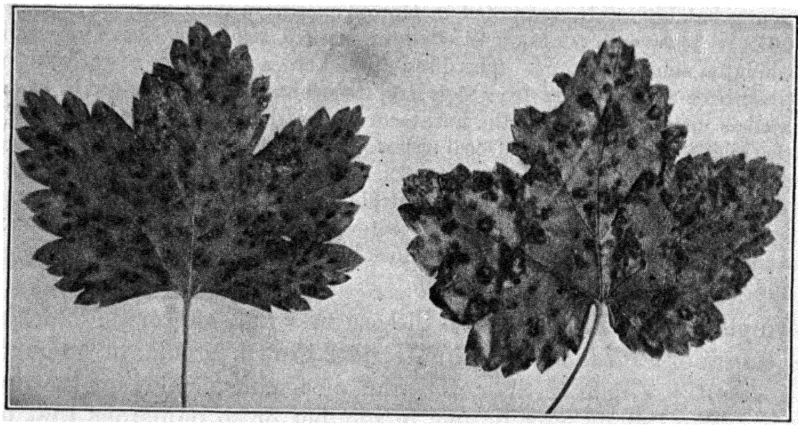

FIG. 153.—Anthracnose (*Pseudopeziza ribis*) on currant leaves.

"The dropping of the leaves so early in the season must seriously interfere with the proper ripening of the wood and the formation of fruit buds for next year" (Stewart), so that the injury is not confined to the season of attack. The injury to the fruit crop in seasons of light attack is probably negligible, but in severe infections heavy losses are experienced. In some recorded cases the yield has been reduced by one-half, while the fruit was also of inferior quality. Severe defoliation affects the development of the fruit, and in hot, dry weather the injury may be increased by sun scald and shriveling of the berries.

Etiology.—Anthracnose is caused by *Pseudopeziza ribis* Kleb., a Discomycete or cup fungus, which produces its ascigerous stage on the old fallen overwintered leaves, and a conidial or Glœosporium stage on the various lesions produced during the growing period. The causal fungus was named *Glœosporium ribis* by Montague and Desmaziers in 1867, and was known under this name until the work of Klebahn in 1906. Klebahn found the apothecial form in the spring on the overwintered

leaves that had been affected with the typical anthracnose lesions, and was able to produce typical lesions upon the growing parts by inoculations with the ascospores. The genetic connection between the ascus and conidial stages was still further substantiated by the production of very characteristic and similar colonies in cultures from both types of spores.

In the parasitic stage of the fungus, typical acervuli are produced on the lesions. The mycelium becomes aggregated into a basal stroma in the center of a leaf spot and spores are formed which finally rupture the

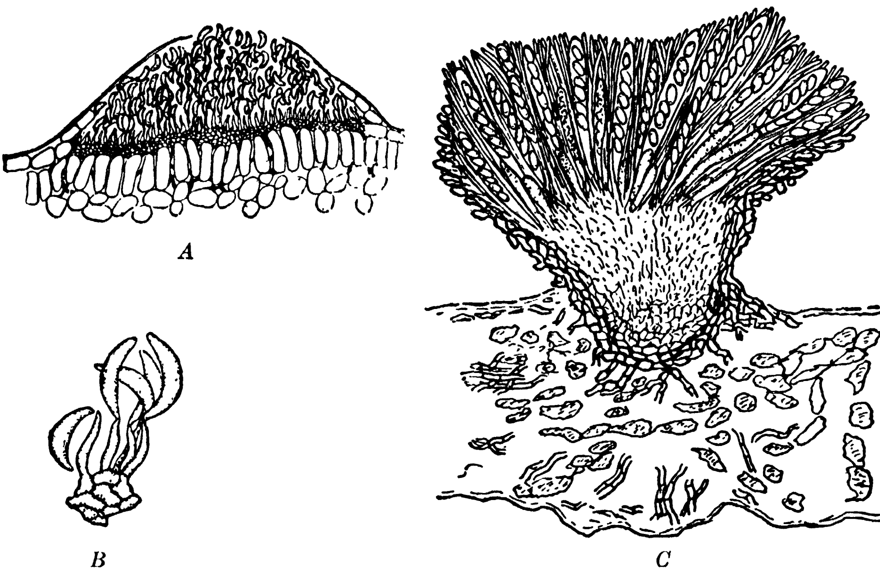

FIG. 154.—*Pseudopeziza ribis.* *A*, section of an acervulus; *B*, small portion of acervulus more highly magnified, showing conidiophores and conidia; *C*, vertical section of an apothecium. (*A*, *after Stewart*; *B*, *after Duggar*; *C*, *after Klebahn*.)

epidermis, and accumulate as the characteristic gelatinous masses described under Symptoms and Effects. The conidia are hyaline, strongly curved or falcate, commonly 19 by 7μ, but varying from 12 to 24 by 5 to 9μ. Since the conidia are embedded in a gelatinous mass they are not wind-disseminated, but are liberated by the dissolving of the gelatinous matrix by rains which carry the spores to adjacent uninfected portions. New infections may thus result during the growing season when moisture conditions are favorable for spore germination. The characteristic lesions may appear after 10 to 14 days. It is stated that conidia produced late in the season may live through the winter, while there is a possibility that the mycelium in the canes may tide the fungus over the winter.

The infected leaves fall to the ground but the mycelium persists and develops as a saprophyte in the dead leaf tissue. This saprophytic

mycelium organizes apothecia which reach maturity in the spring. They appear as minute, fleshy, disk-shaped structures, which break through the epidermis of the overwintered leaves. They remain partially embedded in the leaf tissue and the basal stroma, and the hymenium or ascus layer is in part surrounded by thick-walled cells of the old mycelium. The numerous club-shaped asci, mingled with simple or branched, sometimes one-septate paraphyses, bear eight hyaline, ovoidal spores. When the apothecia mature, the ascospores are forcibly discharged and are carried by the wind to the young leaves, and thus early infections result. The early lesions soon begin the formation of acervuli and from that time on secondary infections result from the numerous conidia.

Exactly what conditions operate to produce an epiphytotic seem uncertain. The disease was very severe in New York in 1889 and in 1901, both seasons being characterized as especially wet. It is significant that anthracnose was epiphytotic in Washington in 1915, and that apple scab was also unusually severe. These observations are opposed to the experience in Bavaria, where the disease was very severe during two rather dry seasons. It would seem that favorable temperature and moisture conditions during the early part of the season are of more influence than abundant rains later in the season.

Host Relations.—Anthracnose occurs on both wild and cultivated species of currants. Black currants (*Ribes nigrum* and *R. aureum*) are generally more resistant than the varieties of red and white currants (*R. rubrum*), although these show varying degrees of susceptibility. Stewart and Eustace report Fay's Prolific and Victoria as very susceptible while V. Stewart adds White Grape. According to Stewart and Eustace, Prince Albert and Pres. Wilder were perfect in foliage when standing side by side with the susceptible varieties, but V. Stewart puts the Wilder in the susceptible list. The disease also occurs on gooseberries, but they are generally more resistant than currants, and are seldom greatly injured. V. Stewart reports the Pearl and Downing as more resistant than Smith, Industry, Whitesmith and Houghton.

The disease is generally reported as more severe on older bushes than on cuttings, but these may become infected if grown in close proximity to old, diseased bushes.

Prevention or Control.—Since the causal fungus overwinters in the fallen leaves, a cultural practice which will bury them or otherwise prevent the formation of the ascus fruits should be of value. In small plantings this might be accomplished by raking out the fallen leaves and burning them, but plowing and clean cultivation are more suited to larger plantings. Pruning to avoid too heavy growth of canes, so that there may be a better circulation of the air and more penetration of light to the lower foliage will help in reducing the severity of infections. Main reliance, however, must be placed on the use of a protective fungicide to prevent the infections: (1) from the spring crop of ascospores produced on

the fallen leaves; and (2) from secondary infections produced during the growing season, from the conidia developed on the leaf lesions. The frequency of severe development of the disease in any environment must be the guide as to whether spraying will be a paying practice.

A number of different fungicides have been tested and have given successful control: (1) Bordeaux mixture, 5–5–50; (2) lime sulphur, 1–40 or 1–50; (3) sulphur-lead mixture consisting of 90 parts finely ground sulphur and 10 parts powdered lead arsenate as a dust. Bordeaux was first recommended by Stewart and Eustace, and later gave good control, according to the experimental tests of Ewert in Germany (1907) and V. Stewart in New York (1915). Lime sulphur 1–40 or 1–50 gave as good control as the Bordeaux, according to V. Stewart (1915), and sulphur dusting was recommended as a result of later tests (1917), especially for nursery stock.

Successful control depends on having the young developing foliage protected with the fungicide previous to the expulsion and dissemination of the ascospores, and upon the continued protection of the foliage during the growing season. The following applications of the selected fungicide should be made: (1) when the leaves are unfolding; (2) after an interval of 10 to 20 days; (3) later sprayings after similar intervals until a maximum of six applications have been made. In some environments or in dry seasons the first two applications will give practical control, while under more humid conditions the larger number of treatments will be required.

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ALFALFA LEAF SPOT

Pseudopeziza medicaginis (Lib.) Sacc.

Alfalfa is affected by a number of parasitic fungi which cause a spotting of the foliage, but the trouble under consideration has been so gener-

ally referred to as the alfalfa leaf spot, that the name has been allowed to stand. This common leaf spot has also been called the leaf rust or blight, and the other spot diseases with which it may be confused are as follows:

Yellow leaf blotch (*Pyrenopeziza medicaginis*)
Ascochyta leaf spot (*Ascochyta spp.*)
Stagonospora leaf spot (*Stagonospora carpathica*)
Cercospora leaf spot (*Cercospora medicaginis*)
Pleosphærulina spot (*P. briosiana*)[1]
Alfalfa rust (*Uromyces striatus*)

The characteristics which will be noted for the true leaf spot should readily distinguish it from the spots caused by the other parasites.

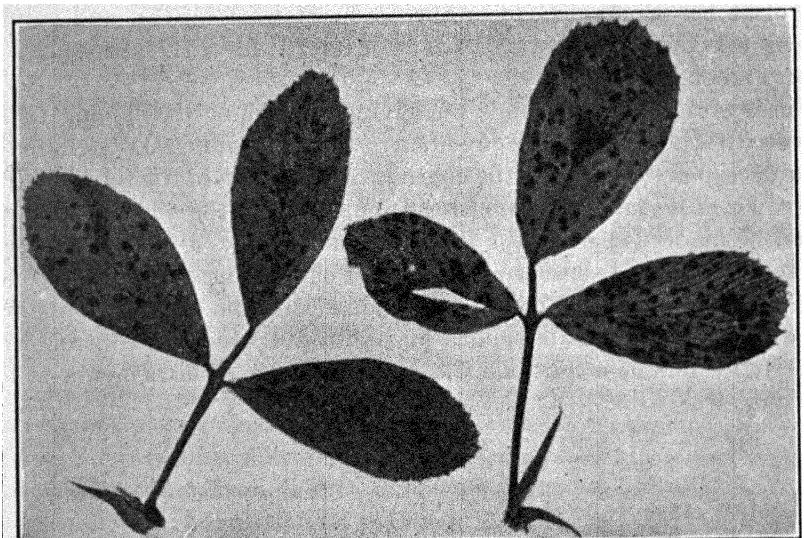

FIG. 155.—Alfalfa leaves showing spotting due to *Pseudopeziza medicaginis*.

History and Geographic Distribution.—What may have been this disease was first recorded in 1832 by Madam Libert on a relative of our cultivated alfalfa (*Medicago lupulina wildenowii*). Most of the early references to the trouble are scattered through the various mycological writings, where principal attention was given to the causal organism. Alfalfa leaf spot was recorded as common in Germany as early as 1869, and was first observed in the United States in 1856 and definitely recorded by Berkeley in 1875. From the simultaneous studies of Voges in Germany and Voglino in Italy (1909), it seems that the disease was general in Europe at that time, but Brefeld had made some detailed studies some years earlier (1891). The first work on the disease in America was published by Chester (1891), who first concluded that it must be seed borne, but his seed-treatment tests failed to substantiate this notion and he then advanced the idea that there must be a general atmospheric infection. The next experimental work of importance was by Combs (1897), who came to the following conclusion:

"First, the plants become affected by the spores carried by the air; and, second, the disease is strictly local, *i.e.*, its mycelium does not affect the stem or root, but is confined to the limited brown spot on the leaf." A very comprehensive study has been

[1] See note on this leaf spot, Chap. XXII.

published by Jones (1919), based on wide observations and extensive laboratory studies. The disease has spread with the cultivation of alfalfa, and can now be found in slight or in severe infestations wherever alfalfa is grown.

Symptoms and Effects.—There are two characteristics of the leaf spot caused by *Pseudopeziza medicaginis* which usually serve to distinguish it from spots caused by other parasitic fungi. The first of these is the circular shape and limited size of the spots. The second is the presence of a small raised disk that appears in the center of the spot when it has reached full development. The edge of the spot may be smooth and definite, especially if the leaf has been much exposed to the sun, or it may be more or less dendritic, with a fringe of olive-colored rays. No marked killing or sinking of the leaf tissue occurs. In size, the spot rarely exceeds 2 or 3 millimeters in diameter (Jones).

The spots are brown or almost black, and the central disk, which is more frequently on the upper surface, may appear as a jelly-like drop or almost as dark as the surrounding portions of the spot, the former appearance being noted under moist, the latter under arid, conditions. The spots may vary in number from a few to a leaflet to a very large number (50 to 100 or more). With moderate infections the average size is 1.5 to 2 millimeters in diameter, but with the very heavy infections the spots may remain as mere specks. In many infections there may be more or less chlorosis of the intervening tissue, with the green color persisting longest around the spots. The extremes of yellowing are likely to occur on the most shaded foliage, even though the infections may not be so numerous as on some leaflets with more exposure. The lesions may also appear on the petioles and succulent stems as elliptical, brown or black spots, 1 to 3 millimeters in length.

The injury from the disease in the established fields is due to the early shedding of the lower leaves, which are the first ones attacked. In severe attacks many leaflets may have fallen to the ground before the time of cutting, while many others which were less affected will shatter off during the curing and handling of the hay. Sometimes the final product from such fields is little more than a mass of naked stems, while the really valuable portion, the leaflets, is left behind in the field. It would seem that heavily infected plants would yield a crop of lower nutritive value exclusive of the loss from shattering. Stewart (1908) reports that, "while it may seriously affect the first cutting in June, the second and third cuttings are the ones most likely to be injured. Overripe plants are especially liable to attack." On the irrigated or upland ranches of the West, where the midsummer rainfall is slight, the first cutting generally shows the maximum infection. Severe cases which have been observed in the middle of a summer drought were probably due to infections which occurred during the previous period of humid conditions.

If for any reason the plant is growing slowly, the stand is thick and the weather is frequently wet, only a few of the upper leaves reach full development

before they are covered with the disease . . . Stands which are growing rapidly keep most of the upper leaves well above the rising invasion of the fungus and show little harm (Jones).

Old-established plants are probably never killed outright by leaf spot, but young seedlings are sometimes completely ruined. In some cases where slightly acid soil has lowered the vigor of the young plants, the disease has been noted in unusually severe form.

It is probable that the average farmer underestimates the amount of damage from this disease. There is, however, a constant toll, small under dry conditions, but larger under more humid conditions, which should not be overlooked. Chester (1891) reported that his experimental plots at the Delaware Station were severely attacked and that some of them were completely destroyed, while Pammel noted losses of 50 per cent in Iowa in 1890. Combs (1897) stated:

There is no doubt that this disease, which attacks the plant at any time after it has made a growth of 4 to 6 inches from the seed, is the principal cause of the non-success or partial success of this most excellent forage plant in this part of the country.

Etiology.—Leaf spot is caused by *Pseudopeziza medicaginis* (Lib.) Sacc., a species of cup fungus which appears to be confined very largely to alfalfa or to other species of Medicago. It was first described (Libert, 1832) as *Phacidium medicaginis* from specimens on *Medicago lupulina wildenowii*, and the fungus found on alfalfa by Desmazieres (1841) was assumed to be identical. In 1883 Saccardo transferred the alfalfa leaf-spot fungus to *Pseudopeziza*, which had been established with *P. trifolii* as the type. For some time after this mycologists believed that the two similar forms on clover and alfalfa were identical, but Jones (1919) has recently presented convincing evidence as a result of pure culture inoculations that the clover and alfalfa species are distinct.

The disk at the center of the leaf spot is the fruiting body or *apothecium* of the pathogene. Mature fruits are 0.5 to 1.5 millimeters in diameter, slightly raised, but sessile and usually surrounded by the torn edges of the leaf epidermis.

The apothecia on alfalfa are usually solitary, except on overwintered leaves. where several clustered apothecia may develop on a stroma. As the hymenial layer develops, the stroma from which it arises becomes thicker, forming in and among the collapsing leaf cells. The epidermis is ruptured, and the hymenium is raised above the surface of the leaf (Jones).

Asci are 60 to 70 by 10μ and the paraphyses continuous, unbranched, swollen at the ends, and slightly longer than the asci. The ascospores are uniseriate or irregularly biseriate, continuous, hyaline, biguttalate, irregularly oval and 10 to 12μ long. *P. trifolii* has slightly larger spores, some of which are slightly flattened on one side.

European writers have reported an associated conidial stage, three different types being described, but Jones has shown that they are the fruits of other fungi. Conidium-like structures are produced on the mycelium in pure cultures, in the form of oval cells, 3 to 8 by 3 to 5μ, from the ends of lateral branches or swollen cells (Jones), but even these are not known to be formed under field conditions, and it is the belief that the fungus relies entirely on the ascospores for dissemination.

Under conditions of suitable temperature and moisture the spores from mature asci are forcibly discharged by the rupture of the ends of the asci, and may be thrown for a distance of several millimeters. They

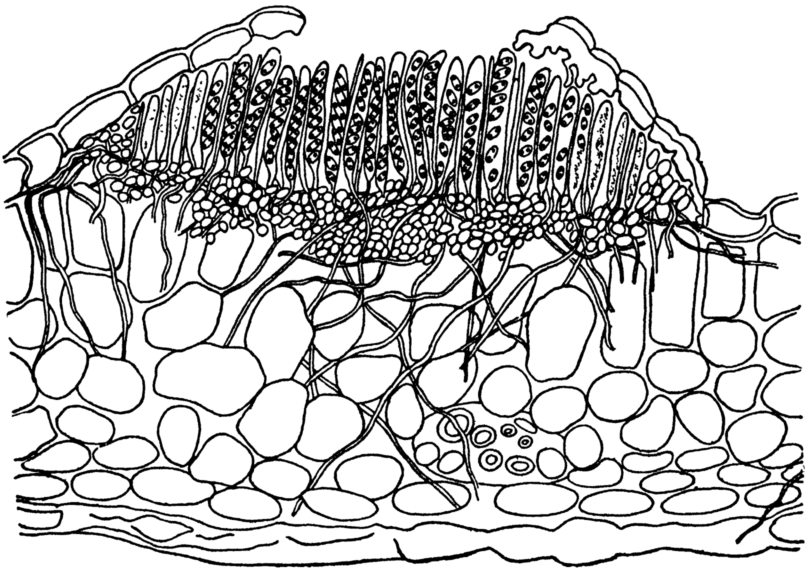

FIG. 156.—Vertical section through an apothecium of the alfalfa leaf-spot fungus (*Pseudopeziza medicaginis*). (*After Combs.*)

stick readily to surfaces with which they come in contact. Some of the fruits will expel their ascospores during the course of the growing season and these will serve for the immediate spread of the disease, while other fruits which are retarded in their development, or form later in the season, will persist on the old fallen leaves and be ready to expel spores in the following spring. New asci may develop in old apothecia and new apothecia may form around the old ones.

Spores expelled naturally from asci are capable of germinating at once upon a moist surface. The entry of the germ tube in leaf infections is not through stomata, as was formerly stated, but

The germ tube emerges from the spore either within or at the margin of the area of contact of the spore with the leaf and passes directly through the cuticle into the epidermal cell. Apparently the germ tube must enter the leaf at the moment of emergence from the spore, if at all. After passing through the wall,

the germ tube quickly expands to normal size. When it reaches the center of the cell it usually divides into two or three branches, which pass into the adjoining epidermal cells or down into the palisade layer (Jones).

There can be little doubt that the new infections are started by wind-borne ascospores.

The ascospores appear to be quite resistant to desiccation. In experimental tests with spores dried on plaster of Paris blocks Jones concluded that drying alone would not kill them in less than 1 year, and that freezing during the drying had no injurious effect. This behavior of the spores is of some importance as indicating the possibility of their survival in viable condition on the surface of overwintered seed. Miss Massee (1914) supports the idea of the disease being seed-borne by microscopic examinations of commercial seed which revealed "the fungus present in abundance on minute fragments of leaves and calyces, and rarely on the seed itself." As a result of tests with treated seed Jones comes to the conclusion that there is little or no evidence that the disease is seed-borne.

In view of the fact that *P. medicaginis* seems confined largely to alfalfa, the probability of the first infections in a new field originating from some other host appears remote. Although positive evidence is lacking, it would seem logical to believe that the fungus may be introduced into new distant localities first with the seed in some form, but that general spread throughout the field or to adjacent fields is accomplished by the wind-borne spores from the first infections.

Control.—In considering the control of this disease the following points should be kept in mind: (1) The ascospores are the only kind of spores which are known to function in nature; (2) these are formed on the leaves and many of the spore-bearing fallen leaves carry the fungus over the winter; (3) the disease does not spread from clover to alfalfa; (4) alfalfa is the principal host in the regions where alfalfa is generally grown; (5) the pathogene is extensively wind-disseminated; (5) seed disinfection does not prevent the disease in new plantings in regions in which alfalfa is already established.

In the light of these facts the only practical measure that is recommended is early cutting. This accomplishes two things: (1) The fungus is prevented from maturing its apothecia; (2) a field is harvested before shedding of leaves has lowered the value of the crop. No exact date of cutting can be specified, but a field which appears to be heavily infected should be carefully watched and mowed before the foliage has begun to drop to any extent.

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CHERRY LEAF SPOT

Coccomyces spp.

This disease of the cherry is characterized by the production of localized dead spots on foliage, fruit and fruit pedicels, the serious aspect being the defoliation which is likely to result. The disease is known by various common names, such as "leaf blight," "leaf spot," "yellows," "yellow leaf" and the "shot-hole disease." The last name has been frequently used because the dead areas often drop out, leaving circular or irregular perforations, while "yellows" and "yellow leaf" have been suggested by the pronounced chlorosis of the foliage which sometimes accompanies severe spotting.

History and Geographic Distribution.—Leaf spot of the cherry was first noted from Europe in 1884 on *Prunus padus*, and according to Sorauer and Lindau is confined almost entirely to that species in Europe. The disease is reported by Aderhold as having been common on both sweet and sour cherries in Europe for 10 years prior to 1901. The related plum leaf fungus was studied by Arthur in New York (1886–1889) and Pammel gave special attention to this "spot disease of cherries" in Iowa in 1891. The causal organism was very imperfectly known until the work of Higgins in 1914, when an ascigerous stage on the overwintered leaves was definitely connected with the conidial stage. Since 1891 special bulletins on either life history or control have been issued from New York (1901, 1914, 1916), Wisconsin (1918), Michigan (1921, 1925), Iowa (1897) and Nebraska (1908) Experiment Stations and by the U. S. Department of Agriculture (1919). According to Roberts and Pierce (1919), "cherry leaf spot occurs quite generally over the eastern half of the United States and has been reported as very destructive in Illinois, Iowa, Nebraska, Michigan, Connecticut, New York and New Jersey." It is common in eastern Canada and has been reported from California, Oregon and Washington. In the West it seems to be confined very largely to the more humid coast sections. It has also been reported from South Africa and other foreign countries.

Symptoms and Effects.—The leaf spot first appears upon the foliage as small, purple or reddish, circular spots, which later enlarge and turn brown. On certain varieties the brown spots may remain surrounded by a zone of reddish brown or in late infections may remain as small purple spots. The spots, ⅛ inch or less in diameter, may be few in number or they may be so numerous as to coalesce and form large irregular dead areas. During humid periods whitish spore masses may appear in the center of the leaf lesions, being much more numerous on the lower than on the upper surface. It frequently happens that the dead brown tissue

of the leaf lesions becomes separated from the surrounding tissue and falls out, leaving characteristic perforations which have suggested the common name of shot hole. When several perforations occur in close proximity they may form irregular or ragged holes. The shot-hole effect seems to be more frequent on sour than on sweet cherries. In severe or later stages of the disease there may be a pronounced chlorosis of the leaf tissue between the lesions, and affected trees may present a striking yellow appearance, hence the name "yellows" or "yellow leaf." Seriously affected leaves may fall prematurely and it is not uncommon for trees to be nearly defoliated by July 1 or a little later. Trees which have had their vitality weakened appear to suffer the most extreme defoliation.

FIG. 157.—Cherry leaves showing spotting due to *Coccomyces hiemalis.*

Especially severe yellowing and defoliation have been noted on English Morello trees which were suffering from a fungous trunk and root rot.

Infections may occur on the fruit and fruit pedicels, and rarely upon the young shoots. The first report of pedicel infection for America was by Stewart and Eustace (1901), who reported one case of English Morello trees in which

> The fruit pedicels were so generally attacked by the fungus that it was somewhat difficult to find one which was entirely free from the brown spots. The spots were from ⅛ to ¼ inch in length and extended one-third to one-half the distance around the pedicel. Often the spots coalesced, and then a large portion, or even all, of the pedicel was brown.

The presence of the pedicel lesions, combined with the defoliation, causes the fruit to ripen unevenly. Direct attacks of the fruit are of minor consequence, but fruit lesions may occur as small brown spots.

The chief injury to the fruit, however, results from the loss of vitality of the tree, due to the loss of leaves. In case of severe attacks the fruit often fails to mature, and wood and bud formation are seriously hindered. Repeated severe attacks may kill the tree (Keitt).

The disease not only affects the crop for the season of attack, but, if severe, it means a "laggard, sour crop of half-sized fruit" for the next year.

In another way the leaf cast works damage to the tree. Winter injury to cherries should be uncommon considering the hardiness of this fruit. It is notorious that orchards where leaf spot has injured the trees suffer most from winter injury. The impoverished limbs and twigs do not possess resistance (Coons, 1921).

Leaf spot was recognized by the earlier studies as a very important disease of nursery stock. Pammel (1891) wrote:

It is, in fact, so bad that the common varieties of the cherry cannot be grown from the pits.

A case is on record where 40,000 young cherry trees were lost on account of leaf blight alone. The loss in Ohio in 1905 is estimated at $25,000. The preceding year it is estimated to have caused a loss of 8 per cent in Maryland. One nursery company in Nebraska claims to have lost $40,000 in 1903 on account of this disease (Hesler and Whetzel, 1917).

The examples serve to show what the disease will do in nursery stock if allowed to develop unchecked.

Etiology.—The leaf spot of cherries is due to two different species of ascomycetous fungi belonging to the Phacidiales: *Coccomyces hiemalis* Higgins, which affects *Prunus avium*, sweet cherries, *P. cerasus*, sour cherries and *P. pennsylvanica* (and also *P. mahaleb*, according to Keitt, (1918*b*); and *Coccomyces lutescens* Higgins, which attacks chokecherries, wild black cherries and the mahaleb cherry (*P. mahaleb*). The fungus was first described from its conidial stage in 1884 as *Cylindrosporium padi* Karst. and, until the work of Higgins in 1914, had been generally known by that name. The similar leaf spot of the plum had also passed under the same name, as they were supposed to be identical. In 1876 Peck described a fungus from the wild black cherries of the Adirondack Mountains as *Septoria cerasina*. This and *Septoria ravenelii* Thüm., with which it seems to be identical, have been shown to be *Cylindrosporium*, the conidial fruit being a typical acervulus. Arthur made a study of the plum leaf fungus and in 1887 described it as *S. cerasina* Pk., but he was apparently dealing with Cylindrosporium. He recognized an ascus stage on the fallen overwintered leaves which he did not name, and which he believed was a stage in the life cycle of the plum fungus, although definite proof was not offered. This discovery of Arthur was apparently overlooked, for Duggar (1909) wrote: "No ascogenous stage of the fungus is known, and there is some doubt as to the ordinary method of wintering over." The detailed studies of Higgins (1914) have shown by cultures

and inoculations that the acervular stage on the living host is but a part of the life cycle of the Ascomycete which produces its apothecial stage on the fallen overwintered leaves. The following description will apply to *Coccomyces hiemalis*, the common species of our cultivated cherries:

The mycelium is intercellular, with haustoria which penetrate the host cells. The haustorium enters through a very small hole in the cell wall and is very much attenuated as it enters, but the end enlarges into an oval elliptical body which contains a nucleus and a comparatively large vacuole. After the haustorium has entered, the protoplasm of the invaded cell often deposits a cellulose sheath around the haustorium, apparently similar to that formed around the haustoria of the Erysiphaceæ as described by Smith (Higgins, 1914).

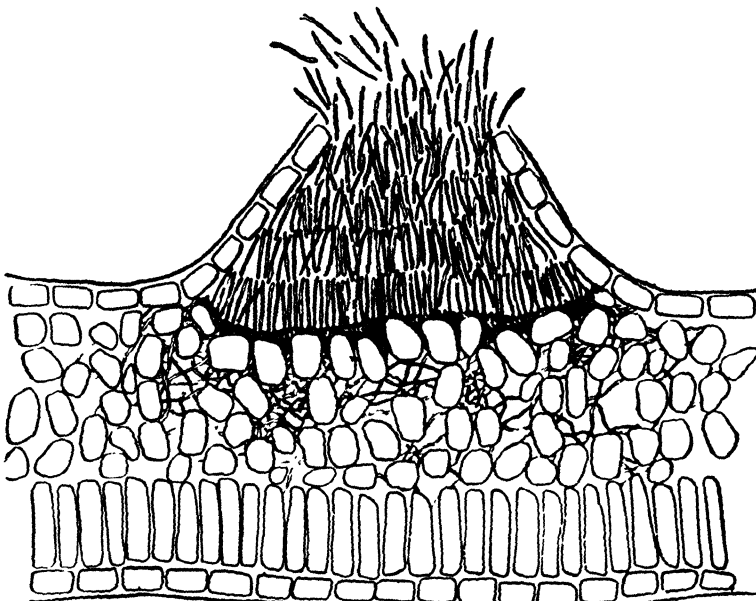

Fig. 158.—Section of an acervulus showing the Cylindrosporium stage of cherry leaf-spot fungus. (*After Stewart, Cornell Circ.* **21**, 1914.)

The amount of killing of the host tissue is exceedingly variable, the minimum being just a few cells in direct contact with the stroma of the acervulus. Apparently no very poisonous toxin or enzyme is produced.

Some lesions fall away before there has been time for the formation of the conidial fruits. Those which do persist sufficiently long generally give rise to one or more acervuli, which appear either above or below. The acervulus consists of a disk-shaped stroma which forms beneath the epidermis, at first only one cell thick, but increasing with age. Conidiospores develop on the upper or outer surface, at first in the center, and then centrifugally. The stroma extends laterally between the epidermis and the mesophyll, and when the conidia have been formed in sufficient number the epidermis is lifted up and ruptured, and they are forced out

upon the surface as yellowish-white or whitish opalescent, sticky masses or sometimes as more elongated tendrils. The conidia are hyaline, elongate, curved or flexuous, 45 to 60 by 2.5 to 4μ and continuous or one- to two-septate.

The production of typical Cylindrosporium conidia (Macroconidia) ceases towards the end of the growing season, and from that time until the leaves fall the same stromata give rise to large numbers of microconidia, 4 to 5 by 1 to 5μ, which are abstricted from the base of short branched conidiophores (these are probably what Arthur in 1886 classed as Phoma spores).

Almost simultaneously with this change in spore form, the stroma begins to develop downward through the mesophyll and palisade layers of the leaf. The stroma usually extends entirely through to the upper epidermis but remains covered both above and below by the leaf epidermis (Higgins, 1914).

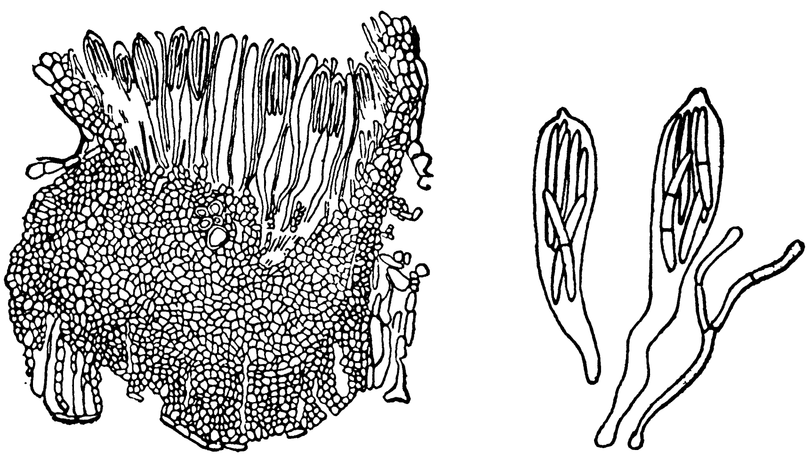

FIG. 159.—*Coccomyces hiemalis.* *A*, section of an apothecium; *B*, asci and paraphyses enlarged. (*After Higgins.*)

This dark stroma begins to swell towards the lower side of the leaf during the first warm spring days, and the hymenial layer soon becomes evident, the paraphyses first, followed by the asci. In April or May the asci enlarge rapidly and raise the covering which breaks in a more or less stellate fashion. Soon after the rupture the apothecia are mature and the ascospores are then forcibly expelled from pores in the papillate apex of the asci. The asci are clavate, 70 to 95 by 11 to 14μ, eight-spored and have a long stout pedicellate base; the hyaline ascospores are fascicled in the large end of the ascus, linear, 30 to 50 by 3.5 to 4.5μ, continuous or one- to two-septate; the paraphyses filiform, septate with apex slightly enlarged and often hooked or forked.

After the ascospores are shed the asci and paraphyses disappear and long slender conidia are formed on short conidiophores which arise apparently as

branches from the base of the paraphyses. They are once or twice septate and resemble Cylindrosporium conidia but are usually longer and a little more slender (Higgins, 1914).

The conidia produced from the acervuli have been shown to lose their vitality rather quickly after drying, but fresh conidia germinate readily under proper conditions of temperature and moisture. During humid periods they are produced in abundance and are washed away by rains or are carried by other natural agencies to healthy foliage and thus serve to spread the disease during the growing season. Apparently the conidia do not live over the winter, and there is but little evidence that any active lesions persist on the young twigs. The ascus fruits are undoubtedly the source of the first leaf infections of the season, and the wind-borne ascospores are set free shortly before the first leaf lesions appear. It has been pointed out that they are more active in producing infections than the Cylindrosporium conidia. The conidia produced in the apothecia following the liberation of the ascospores are also capable of causing infections, but it is uncertain how important a part they play in the life of the parasite. The microconidia or spermatia are not known to be functional in producing new infections.

Coccomyces species can be grown in cultures from either conidia or ascospores, but their behavior in culture is variable. The following statement of cultural characters is based on the work of Higgins (1914).

Growth from the conidia is very slow, the colonies being visible to the naked eye only after 10 to 15 days. They show then as small whitish specks, which consist of stromata covered with conidia similar to those formed on the leaf lesions. After about 2 months a colony enlarges to a hemispherical mass, 0.5 to 1 centimeter in diameter, and is black and crust-like in *C. prunophoræ* from plums, dark but not crust-like in *C. hiemalis*, but creamy white and more floccose in *C. lutescens*. The dropping out of the circular leaf lesions producing the shot-hole effect is due to the enlargement of a layer of cells at some distance from the ends of the mycelium. Their enlargement is so abrupt and so great that the active cells separate from the adjoining inactive cells inside (Higgins).

The separated leaf tissue "turns yellow, shrinks rapidly and soon drops out." It is believed that the enlargement of the cells is due to increased osmotic pressure, and that the production of shot holes is correlated with the amygdalin content of the leaves. According to this theory the amygdalin is broken down by an enzyme which is set free when the affected tissue wilts, and substances are produced which increase the osmotic pressure. This theory is substantiated by the fact that amygdalin is not found in *Prunus avium*, and that shot-hole formation is rare in this species. According to Cunningham (1928), "a definite cicatrice is formed about the edge of the lesion, thus isolating the diseased portion from the healthy." The three species of Coccomyces produce similar effects.

Host Relations.—As previously noted, two species of Coccomyces affect cultivated and wild cherries, while one species, *C. prunophoræ*, affects plums. Since the work of Higgins (1914) extensive cross-inoculation tests with *Coccomyces spp.* have been made by Keitt (1918*b*). The strains studied were "tentatively grouped as follows, according to the hosts from which they were procured: (1) *P. cerasus*, *P. avium*, *P. mahaleb* and *P. pennsylvanica;* (2) *P. domestica;* (3) *P. virginiana;* and (4) *P. serotina.*" Neither sweet nor sour cherries were infected from strains obtained from wild black cherries or from chokecherries, but a

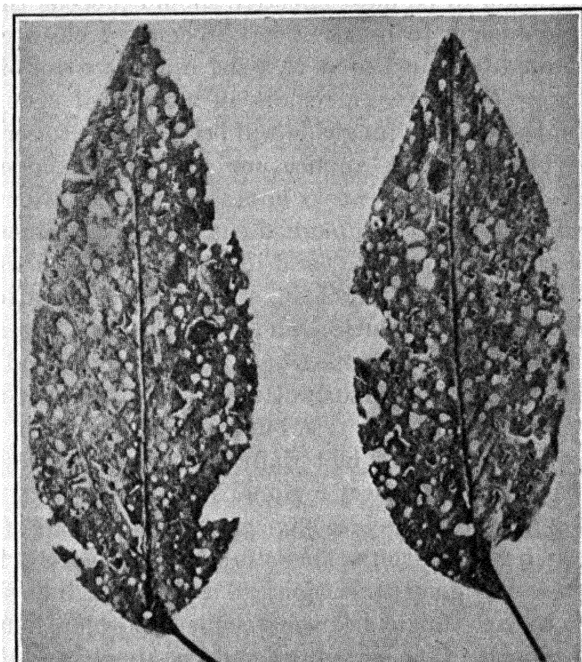

FIG. 160.—Plum leaves showing an extreme case of shot hole due to *Coccomyces prunophoræ.*

limited transfer from the bird cherry (*P. pennsylvanica*) to the cultivated species was noted. The conclusion may be drawn that there is but little danger of the infection of cultivated cherries by Coccomyces species from wild hosts. Conditions in the Inland Empire of the Pacific Northwest offer similar evidence without the use of extensive cross-inoculations. Coccomyces is very common on the native chokecherry (*P. demissa*), but both sour and sweet cherries always remain free from infection even though grown in many cases in close proximity to severely infected chokecherries.

Prevention and Control.—A consideration of the life history of the pathogene will at once suggest two possible lines of procedure: the elimination of the apothecia, or the overwintered fruits, as a source of the first

spring infections; and the protection of the developing foliage by the application of a fungicide.

1. *Early Clean Cultivation.*—If all the dead leaves could be destroyed and if the fungus harbored on no other plants, spraying would not be necessary. While it is not practicable to practice sanitation with such thoroughness as to make spraying unnecessary, it is possible greatly to strengthen the spray schedule by turning under as many of these leaves as is feasible by clean cultivation in the spring before the spores of the fungus are discharged (Keitt, 1918).

Clean cultivation to be most effective should precede the time when the blossom buds begin to break open. Whenever clean cultivation is practiced, it should be timed so as to assist in the control of leaf spot.

2. *Spraying or Dusting.*—In regions in which leaf spot is prevalent in serious form, the use of a fungicide will be necessary to secure commercial control. The following applications are recommended: (*a*) soon after the petals fall; (*b*) 2 to 3 weeks later; and (*c*) just after the fruit has been harvested. Excellent commercial control was obtained in Wisconsin in some seasons with only (*a*) and (*b*) in combination with thorough early clean cultivation. In Michigan, spraying 4 weeks after the petals fall is also recommended (Dutton and Wells, 1925). Spraying before blooming gives no increased protection. The safety period for the first spraying, which is the most important one, may extend through about a week following the fall of the petals, but in unusually warm weather this period for effective spraying is materially shortened. The spraying program should be modified to meet regional variations.

The following liquid fungicides have given good control: (*a*) Bordeaux mixture, 3–3–50 or 2–2–50, the weaker strength for regions of light infestation or if supported by good sanitation; (*b*) commercial lime sulphur 1–30, 1–40 or 1–50. In each fungicide arsenate of lead powder should be added, 1 pound to 50 gallons. The spraying should be timely and thorough and an effort should be made to cover both upper and under surfaces of the leaves. The 1–30 lime sulphur may be used on sour cherries without danger of injury, but 1–40 has proved effective. 1–50 lime sulphur should be used on sweet cherries, according to Roberts and Pierce (1919), but Bordeaux should not be used on these varieties because of the danger of injury. It is claimed that lime sulphur 1–50 may be used with safety by the addition of ¼ pound of iron sulphate to each 50 gallons, which will increase the adhesiveness and lessen the burning properties. Lime sulphur, 3 gallons to 100, is recommended in Michigan (Dutton and Wells, 1925) instead of Bordeaux because of severe foliage injury and serious reduction in the size of the fruit with the latter. Good control, but less effective than with Bordeaux, has been reported from the use of flotation sulphur, 6–50, the three brands Thylox, Ferrox and Gray giving about equal protection (Smith, 1930).

Dusting with sulphur has been recommended especially for nursery stock, but in some cases it has not given adequate control of the disease in orchard trees. A formula frequently recommended is 90 parts of dusting sulphur to 10 parts of finely powdered lead arsenate. The first application in nursery stock should be made when the cherry buds are 8–12 inches high, and whether spraying or dusting is practiced the applications should follow at intervals of about 2 weeks. The serious nature of the disease on nursery stock will necessitate from five to seven applications to give adequate protection.

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IMPORTANT DISEASES DUE TO CUP FUNGI AND ALLIES

I. Helvellales

Root rot of conifers, especially seedling trees (*Rhizina inflata* (Schäff.) Sacc.).—Weir, J. R.: Observations on *Rhizina inflata*. *Jour. Agr. Res.* **4**: 93–95. 1915. Van der Lek, H. A. A.: *Rhizina inflata*, ein Wurzelschmarotzer von Koniferen. *Tijdschr. over Plantenziekt.* **23**: 181–194. 1917.

II. Pezizales

1. *Helotiaceæ:*

Brown rot of stone fruits (*Sclerotinia spp.*).—(See special treatment.)

Drop of lettuce (*Sclerotinia sclerotiorum* (Lib.) Mass. = *S. libertiana* Fckl.).—Causes also wilt or stem rot of various garden vegetables and the cottony rot of lemons. Smith, R. E.: Botrytis and Sclerotinia: Their relation to certain plant diseases and to each other. *Bot. Gaz.* **29**: 369–407. 1900. Stevens, F. L.: A serious lettuce disease. *N. C. Agr. Exp. Sta. Bul.* **217**: 1–21. 1911. Krout, W. S.: Control of lettuce drop by the use of formaldehyde. *Jour. Agr. Res.* **23**: 645–654. 1923. Wakefield, E. M.: On the names *Sclerotinia sclerotiorum* (Lib.) Massee, and *S. libertiana* Fckl. *Phytopath.* **14**: 126–127. 1924. Smith, C. O.: Cottony rot of lemons in California. *Cal. Agr. Exp. Sta. Bul.* **265**: 237–258. 1916. Davis, W. H.: Drop of Chinese cabbage caused by *Sclerotinia sclerotiorum* (Lib.) Massee. *Phytopath.* **15**: 249–259. 1925.

Drop of lettuce (*Sclerotinia minor* Jagger).—Also affects celery and other garden crops. At first confused with *S. libertiana* but now known to be a distinct species. Jagger, I. C.: *Sclerotinia minor*, n. sp., the cause of a decay of lettuce, celery and other crops. *Jour. Agr. Res.* **20**: 331–333. 1920. Beach, W. S.: The lettuce "drop" due to Sclerotinia minor. *Pa. Agr. Exp. Sta. Bul.* **165**: 1–27. 1921.

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Hard rot and tip blight or cotton-ball of the cranberry (*Sclerotinia oxycocci* Wor.).—Shear, C. L.: Cranberry diseases and their control. *U. S. Dept. Agr., Farmers' Bul.* **1081**: 10–11. 1920. Also *U. S. Dept. Agr. Tech. Bul.* **258**: 9–10; 42–43. 1931.

Canker of larch (*Dasyscypha calycina* (Schum.) Fckl.—Hiley, W. E.: The fungal diseases of the common larch, pp. 16–79. Clarendon Press, Oxford. 1919. Meiniecke, E. P.: The European larch canker. *Mo. Bul. Cal. Dept. Agr.* **19**: 506–509. 1930.

White-pine canker (*Dasyscypha fusco-sanguinea* Rehm.).—Stillinger, C. R.: *Dasyscypha fusco-sanguinea* Rehm on western white pine. *Phytopath.* **19**: 575–584. 1929.

2. *Mollisiaceæ:*

Leaf spot of alfalfa (*Pseudopeziza medicaginis* (Lib.) Sacc.).—(See special treatment p. 545.)

Currant anthracnose (*Pseudopeziza ribis* Kleb.).—(See special treatment p. 540.)

"Roter Brenner" of grape (*Pseudopeziza tracheiphila* MT.).—MÜLLER-THURGAU, H.: Der rote Brenner des Weinstocks. *Centralbl. Bakt. u. Par.*, II Abt. **38**: 586–621. 1913. ZILLIG, H. AND NIEMEYER, L.: Beiträge zur Biologie und Bekämpfung des Rotenbrenners des Weinstocks. *Arb. Biol. Reichanst. Forstw.* **17**: 1–65. 1929.

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3. *Cenangiaceæ:*

Twig blight of pine and fir (*Cenangium abietis* (Pers.) Duby).—FINK, BRUCE. Injury to *Pinus strobus* caused by *Cenangium abietis*. *Phytopath* **1**: 180–183. 1911. WEIR, J. R.: Notes on *Cenangium abietis* (Pers.) Rehm on *Pinus ponderosa*. *Phytopath.* **11**: 166–170. 1921. LIESE, J.: Neue Beobachtungen ueber *Cenangium abietis*. *Zeitschr. Forst-u. Jagdw.* **54**: 227–229. 1922.

III. PHACIDIALES

1. *Phacidiaceæ:*

Black leaf spot of arbor vitæ (*Keithia thujina* Dur.).—WEIR, J. R.: *Keithia thujina* the cause of a serious leaf disease of western red cedar. *Phytopath.* **6**: 360–363. 1916.

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Anthracnose of poplar (*Trochila populorum* Desm.).—The conidial form is *Marssonina castagnei* (D. & M.) Sacc. EDGERTON, C. W.: *Trochila populorum* Desm. *Mycologia* **2**: 169–173.

Tar spot of maple (*Rhytisma acerinum* (Pers.) Fr.).—CIFERRI, R.: Un intense attacco del *Rhytisma acerinum* (Pers.) Fr. alle folgie d'*Acer campestre*. *Riv. patol. veg.* **11**: 93–95. 1921. JONES, S. G.: Life history of *Rhytisma acerinum* (Preliminary account). *Ann. Bot.* **37**: 731–732. 1923. BRACHER, R.: Notes on *Rhytisma acerinum* and *Rhytisma pseudoplatani*. *Trans. Brit. Myc. Soc.* **9**: 183–186. 1924. JONES, S. G.: Life history and cytology of *Rhytisma acerinum* (Pers.) Fries. *Ann. Bot.* **39**: 41: 75. 1925.

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IV. Hysteriales

1. *Hypodermataceæ:*

Leaf cast of larch (*Hypodermella laricis* Tubeuf).—Schmitz, H.: Leaf cast of *Larix occidentalis* by *Hypodermella laricis* Tubeuf in North Idaho. *Phytopath.* **13**: 505–506. 1923.

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CHAPTER XXI

DISEASES DUE TO POWDERY MILDEWS AND ALLIES

ERYSIPHACEÆ AND RELATED FAMILIES

A number of families of related ascomycetes showing primitive ascus fruits on the one hand and on the other well-developed perithecia, which suggest either apothecial fruits or typical ostiolate fruits of the Sphæriales or sphere fungi, may be briefly characterized:

1. Aspergillaceæ.—The ascocarps are spherical or tuber-shaped, sclerotia-like, small, firm, membranous or fleshy, without an ostiole and break open irregularly at maturity. The asci are spherical or pyriform and bear two to eight continuous spores. The ascocarps are rarely formed, but the conidial stage is conspicuous and produces many spores. There are but two genera that merit special mention, and these are readily distinguished by their conidial forms:

Penicillium.—Conidiophores erect, septate and branched, with the branches upright or nearly parallel, making pencil-like or brush-like tufts, with the terminal branches or sterigmata bearing chains of one-celled spores which readily separate. In certain species the conidiophores may be grouped into coremia (see also Imperfect Fungi).

Aspergillus.—Conidiophores erect, generally non-septate and terminated by a bulbous enlargement with radiately arranged, simple or branched sterigmata bearing one-celled spores as in Penicillium.

2. Plectodiscellaceæ.—The ascocarps are stromatic, indefinite, innate in the substratum and composed of a basal colorless parenchymatous or prosenchymatous portion in which the globular asci are irregularly arranged, with superficial, darker-colored cells.

Plectodiscella.—Ascospores elongated, transversely three-septate and hyaline.

3. Perisporiaceæ.—Mycelium generally dark, sometimes hyaline when young or within the substratum. Perithecia superficial, dark, without ostiole, and without differentiated appendages. Conidial stages various but never of the Oidium form. Three genera furnish species of economic importance:

Thielavia.—Perithecia globular, brown, completely closed and without appendages. Asci evanescent, spores unicellular and lenticular. Conidia of two types: (1) hyaline *endospores* produced internally in chains in terminal branches or groups of branches, and set free by the rupture of the branch tip; and (2) dark-colored, thick-walled *chlamydospores* borne in chains on hyaline lateral branches, but separating at maturity.

Cleistothecopsis.—Perithecia superficial, irregularly globular, dark brown or black and without ostiole; wall pseudoparenchymatous, outer cells dark, inner hyaline. Asci evanescent, eight-spored, ascospores muriform, dark. The conidial form of the single species has been known under the following names: *Vermicularia circinans, Volutella circinans* and *Colletotrichum circinans.*

Meliola.—Mycelium superficial, dark, making a sooty coating. Perithecia globose, with simple or branched appendages. Ascospores oblong, two- to five-septate or rarely muriform. Conidia of several types.

4. Erysiphaceæ.—Mycelium mostly superficial, hyaline. Perithecia dark, without ostiole, but with characteristic appendages. Asci one to several, two- to eight-spored. Conidial stage referable to the form genus Oidium. This family comprises the true powdery mildews (see more detailed account).

5. Microthyriaceæ.—Mycelium superficial or subcuticular, dark. Perithecia superficial or innate, mostly shield-shaped, dark and radiate, generally with only the upper half well developed, opening by an ostiole or by the rupture of the shield. There is one important genus.

Diplocarpon.—Perithecia spherical to disciform, on a stromatic base, opening by the rupture of the covering membrane or shield, and when mature discomycetous in appearance. Ascospores unequally two-celled, hyaline. Conidia two-celled, hyaline, in an acervulus (Marsonina type).

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THE POWDERY MILDEWS (Erysiphaceæ)

The powdery mildews are obligate parasites which live for the most part on the surface of host parts making first a delicate, hyaline, cobweb-like growth of mycelium (in a few cases the old mycelium may become brown), which soon assumes a white, powdery or dusty appearance due to the development of numerous conidia. It is this feature which has suggested the common name of the family. While the powdery mildews are mainly leaf parasites, they may grow upon stems, flower parts or fruits. Some of the species cause little or no apparent injury to their hosts, while others may cause destructive diseases.

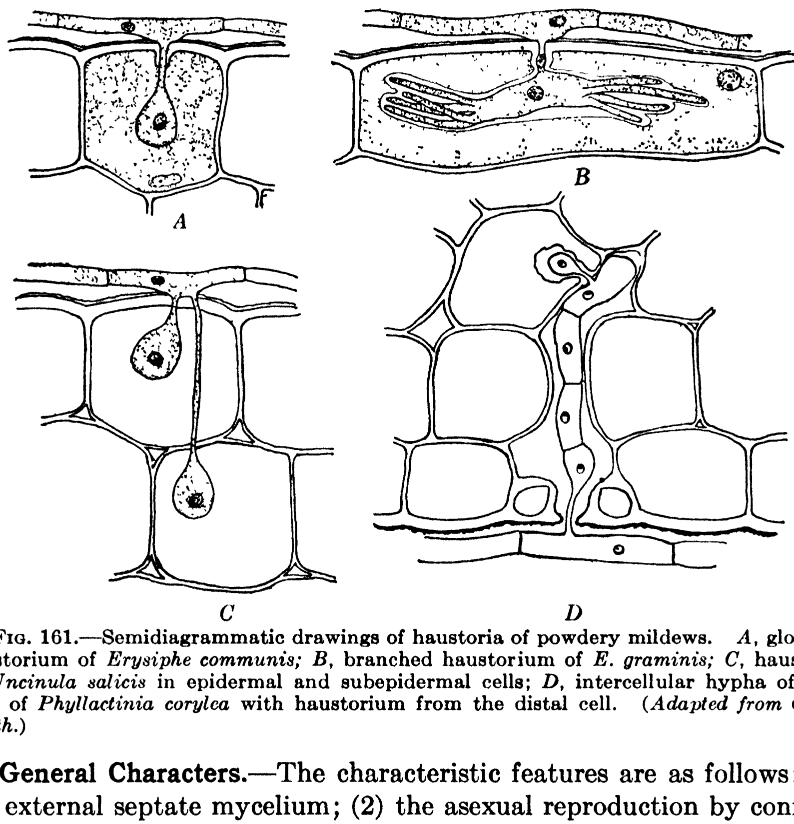

FIG. 161.—Semidiagrammatic drawings of haustoria of powdery mildews. *A*, globular haustorium of *Erysiphe communis; B*, branched haustorium of *E. graminis; C*, haustoria of *Uncinula salicis* in epidermal and subepidermal cells; *D*, intercellular hypha of four cells of *Phyllactinia corylea* with haustorium from the distal cell. (*Adapted from Grant Smith.*)

General Characters.—The characteristic features are as follows: (1) the external septate mycelium; (2) the asexual reproduction by conidia, generally formed in chains on erect unbranched conidiophores; and (3) the sexual reproduction by oögonia and antheridia, with the formation of closed spore fruits, or perithecia, bearing characteristic outgrowths or appendages.

Hyphæ and Mycelium.—While in most species of powdery mildews the mycelium is superficial, the hyphæ form special sucking organs, or *haustoria*, which penetrate the epidermal cells or even into the subepidermal cells for the withdrawal of the necessary food. These haustoria are of

two general types: (1) globular or pyriform enlargements borne by slender penetrating hyphæ; and (2) branched enlargements which very greatly increase the absorbing surface over that afforded by the globular types. In the formation of a haustorium a localized swelling of the host epidermal wall precedes the pushing out of a delicate penetrating hypha. The swollen wall may be pushed in some distance by the penetrating hypha, and may finally be ruptured and persist as a collar around the base

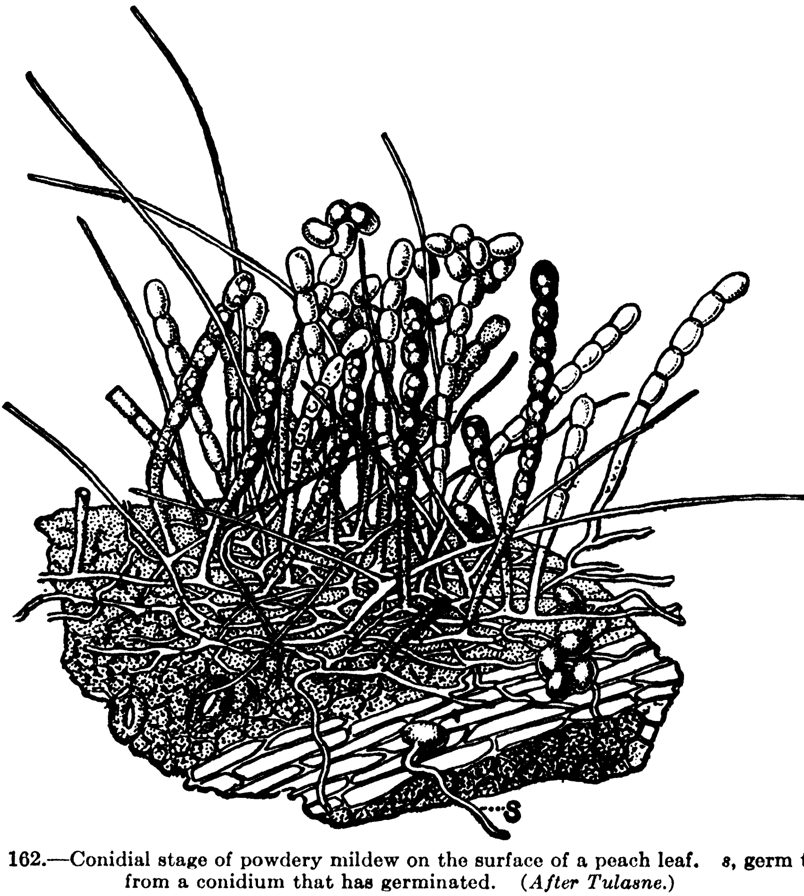

FIG. 162.—Conidial stage of powdery mildew on the surface of a peach leaf. *s*, germ tube from a conidium that has germinated. (*After Tulasne.*)

of the haustorial filament or be carried into the cell as a cellulose capsule surrounding the globular enlargement of the haustorium. In some species the hyphæ form special enlargements or *appressoria* which appear to fix the hyphæ more firmly to the epidermal walls of the host, and in such cases the haustoria are developed from the faces of the appressoria in contact with the epidermal wall. Within the family there is a tendency to the development of internal parasitism. For example, in *Phyllactinia corylea,* the common tree mildew, the haustoria do not penetrate the epidermal cells, but special septate branches grow through the stomata

into the substomatal chambers and then haustoria which enter adjacent cells are formed by these internal branches. One species (*Oidiopsis* (*Erysiphe*) *taurica*) develops an internal mycelium which emerges and forms a surface growth previous to the organization of the spore fruits.

Conidia and Types of Conidiophores.—The common type of conidial formation is by the production of rows of uninucleate, unicellular, oval or barrel-shaped hyaline cells in chains on the ends of erect conidiophores. These conidia are detached and, accumulating on the surface of the host, give the characteristic powdery coating. If heavily mildewed shoots are shaken these spores will fly away in a visible cloud. Before the relation of the conidial stage to the ascigerous stage was understood, the generic name, Oidium, was applied to the conidial stage of the powdery mildews.

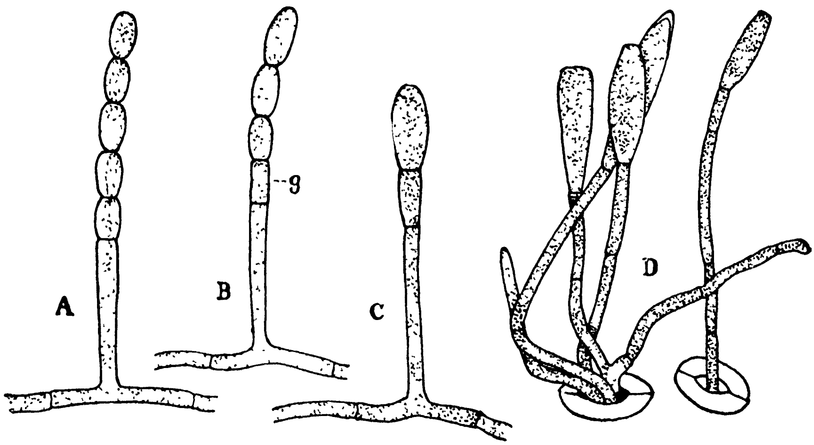

FIG. 163.—Types of conidiophores and the development of conidia. *A*, the conidiophore is the generative cell; *B*, with a special generative cell (*g*) at the base of the chain of conidia; *C*, with a single terminal conidium; *D*, simple or branched conidiophores (Oidiopsis type) from an internal mycelium. (*D*, *after Foëx.*)

This is still spoken of as the Oidium stage, and when perithecia are unknown the mildew may be referred temporarily to the form genus. Four different types of conidiophores are recognized by Foëx (1923): (1) the basal cell is at the same time the pedicel and the generative cell and gives rise to a chain of conidia; (2) a unicellular pedicel bears one generative or mother cell, above which a long or short chain of conidia develops; (3) the conidiophore consists of a row of cells slender below and broader above, which bears a single apical conidium; and (4) the multicellular pedicel, or base, originates from endophytic hyphæ and from this spore-bearing branches arise, and emerge through the stomata of the host, each producing a single conidium. The conidia are capable of immediate germination but are relatively short-lived. They are disseminated by the wind and other agents, but heavy precipitation seems to be unfavorable for their spread, since they may be washed down to the ground and never reach host surfaces on which a new mycelium may be established.

The Spore Fruits or Perithecia.—After a period of vegetative activity and the production of conidia, the fungus forms spore fruits or perithecia, which appear as minute black dots seated in the superficial mycelium. These perithecia, which are formed from June and July to the end of the growing season, are at first hyaline like the mycelium, but later they become a clear yellow and when mature are either a dark brown or black. In specimens collected at the right time, hyaline, yellow and brown perithecia may be found side by side on the same superficial mycelium. Some species of powdery mildews when growing on certain hosts or under the influence of certain environmental conditions produce perithecia but rarely. In some species the development of perithecia takes place only on certain organs of the host, the mycelium on the other parts producing conidia only.

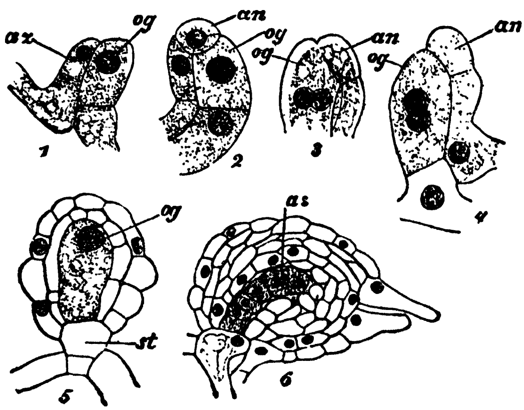

FIG. 164.—*Sphærotheca castagnei.* Fertilization and development of the perithecium 1, oögonium (*og*) with the antheridial branch (*az*) applied to its surface; 2, separation of the antheridium (*an*); 3, antheridial and oögonial nuclei have met; 4, union of the nuclei; 5, fertilized oögonium surrounded by two layers of hyphæ derived from the stalk cell (*st*); 6, later stage in the development of the perithecium. The cell with the two nuclei (*as*) gives rise to the ascus. (*After Harper.*)

A mature perithecium of a powdery mildew consists of a globular or slightly flattened, closed body, 50 to 350μ in diameter, which encloses one to several subglobular to pyriform spore sacs or asci, each bearing 2 to 8 unicellular hyaline ascospores. Flexuous or rigid, simple or characteristically branched outgrowths from the surface cells of the perithecial wall constitute the so-called *appendages.* The origin and the development of a perithecium may be outlined as follows: a lateral branch of a hypha is separated by a cross-septum and enlarges to form an oval, elongated or slightly twisted, uninucleate cell, the *oögonium;* an adjacent hypha produces a branch which remains more slender, and cuts off a terminal smaller, uninucleate cell, the *antheridium,* from the more elongated pedicel cell. Antheridium and oögonium come into contact, and solution of a portion of the separating walls takes place, thus bringing the cytoplasm

of the two cells in communication. The antheridial or male nucleus then migrates into the oögonium and fuses with the oögonial or female nucleus, the fusion constituting the process of fertilization. Following fertilization two changes are initiated which lead to the development of the perithecium: (1) division of the fertilized egg cell to produce a cell aggregate from which one or more asci arise; and (2) the production of hyphal cells from the stalk of the oögonium which grow up around the segmented egg cell and form the enclosing wall of the perithecium.

In the simplest and most primitive of the powdery mildews, as may be illustrated by Sphærotheca species, the fertilized egg cell divides to produce a stalk cell and an apical cell. The former remains without further division, while the latter undergoes division and produces several cells, one of which is binucleate and develops into an ascus. In the forms with several asci the process is very similar, but slightly more complex, the end result being a multicellular aggregate from which the several asci originate.

The wall of a developing perithecium consists of inner and outer layers, the inner consisting of hyaline cells, with abundant cytoplasm, and thin unmodified cell walls; the outer of cells with scanty contents and modified walls which first become yellow, then dark brown. The inner layers occupy all the space not required by the developing asci, supply them with food and are dissolved and appropriated by the time the perithecium is mature. The appendages which are outgrowths of the surface cells of the perithecial wall are of the following types: (1) flexuous, hypha-like and generally unbranched, as in Sphærotheca and Erysiphe; (2) rigid and straight (rarely unbranched), but generally two to several times dichotomously branched, as in Podosphæra and Microsphæra; (3) straight and spirally inrolled or hooked at the apex, as in Uncinula; and (4) rigid and straight, with bulbous base and more slender pointed extremity, as in Phyllactinia.

The perithecia show either a radial or a dorsiventral structure. In some species they remain attached to the substratum, as in nearly all species of Sphærotheca and Erysiphe, while in nearly all others they are provided with some device for detaching them from the substratum. In Microsphæra, for example, the cells on the lower side of the perithecium are thinner walled and larger than on the dorsal or upper side. When these perithecia dry out, the walls of the under sides become concave and separate from the mycelial threads by which they were attached, thus freeing them from the substratum. The loosened perithecia may be detached singly or in groups which are held together by the interlocking appendages and the groups may be washed down by rains or carried away by the wind. They may be again fixed to a substratum by the mucilaginous character of the hyphæ and appendages when acted upon by moisture. The perithecia of Phyllactinia are set free in an entirely different

way. With alternate drying out and absorption of moisture the perithecia are raised and lowered by the movements of the appendages. With loss of moisture the appendages are turned downward, thus raising the perithecium on stilt-like legs, but with moistening the appendages are straightened. This movement is dependent on the structure of the bulbous bases of the appendages, which are thick-walled above and thin-walled below. In this genus the upper surface of the young perithecium is furnished with a ring of penicillately branched appendages which break down into a gelatinous cap about the time the spores are mature. By means of this sticky cap the detached perithecium may adhere upside down to any new surface with which it is brought in contact.

The perithecia in most cases are overwintering fruits, the ascospores being able to germinate only after being exposed to winter temperatures. In a few species the ascospores may be ejected in the fall under favorable conditions of moisture and temperature and germinate at once, but the general period for ascospore expulsion is during the warm spring rains. The perithecium is ruptured by the absorption of water and the swelling of the asci, which protrude through the rupture, and spore expulsion takes place by the successive explosion of the asci. From this behavior it seems that the ascospores are adapted for wind dissemination as in the majority of other ascomycetes. In many species of powdery mildews it is these wind-blown ascospores which are responsible for the first spring infections, since the conidia are short-lived and are not ordinarily able to survive the winter. The first mycelia from the ascospores soon produce conidiophores and conidia which are responsible for the rapid summer spread of the mildew. It is known that some powdery mildews, especially certain species attacking woody hosts, can survive without the production of perithecia. The grape mildew (*Uncinula necator*) was known in Europe for 47 years before any perithecial formation was observed. The oak mildew became generally prevalent in England and on the Continent about 1904, but perithecia were never found until 1911. Even in certain species in which perithecia are commonly formed, it is the belief that they play a minor part in carrying the fungus over the winter. In such cases the dormant mycelium either hibernates in the winter buds and resumes activity when buds open in the spring, or special cells are formed which are highly resistant.

Biological Species.—The existence of specialized physiological strains or so-called biological species within certain morphological species has been demonstrated by means of inoculation tests. For example, the grass mildew (*Erysiphe graminis*) was first shown by Marchal in 1902 to consist of seven physiological strains, and the extreme specialization of this and other mildews has been shown by later studies by Neger, Salmon and Reed. One strain can infect oats and *Arrhenatherum elatius*, but not other grasses; another certain species of Bromus only; another wheat and

related species of Triticum; while still others affect barley and rye and various groups of wild grasses. The occurrence of physiological specialization has been shown for a number of other species of mildews, especially the cucurbit mildew (*Erysiphe cichoracearum*), the pea mildew

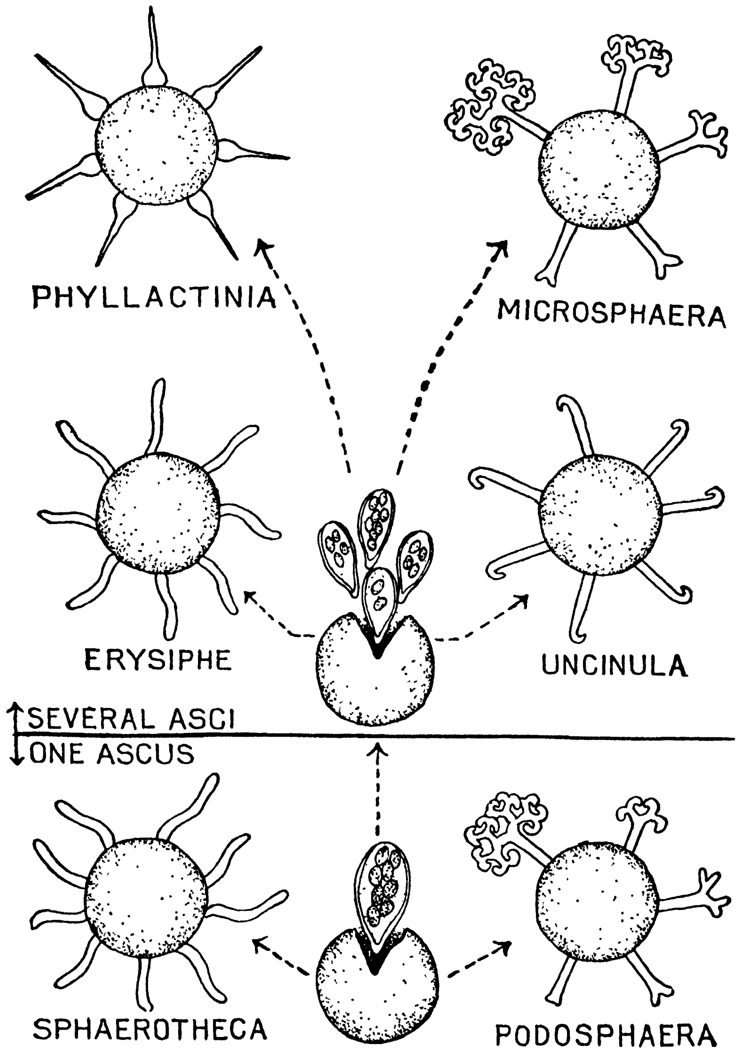

FIG. 165.—Semidiagrammatic drawings of the perithecia of the principal genera of powdery mildews.

(*E. polygoni* with 26 *formæ speciales* according to Hammarlund (1925)), and the rose mildew (*Sphærotheca humuli*). The occurrence of so-called "bridging species" has also been noted. For example, a certain strain may affect host *a* and *b* but not *c*, but later the mildew from host *b* may then be carried over to host *c*. Host *b* may thus act as a bridge by which

the mildew may pass from its original host to a species that was immune to direct attack. The extent to which this is true remains for further investigations to establish.

Classification.—The principal characters on which the genera are based are the number of asci in the perithecium, the symmetry of the perithecia, the kind of appendages and the location of the mycelium.

Subfamily Erysipheæ

Mycelium superficial, sending haustoria into the epidermal cells or into subepidermal cells; asci one or several; appendages various; perithecia radial or dorsiventral, and persisting or becoming detached from the host surface at maturity. The following genera may be briefly characterized:

Sphærotheca.—One ascus; appendages flexuous, hypha-like and generally unbranched; perithecia radial, persisting.

Podosphæra.—One ascus; appendages rigid, dichotomously branched or more rarely simple: perithecia dorsiventral, becoming detached.

Erysiphe.—Several asci; appendages flexuous, hypha-like and generally unbranched; perithecia radial (except *E. graminis*), persisting.

Trichocladia.—Several asci; appendages flexuous and simple or dichotomously branched; perithecia dorsiventral, becoming detached.

Microsphæra.—Several asci; appendages rigid, several times dichotomously branched or rarely simple; perithecia dorsiventral, becoming detached.

Uncinula.—Several asci; appendages rigid, simple or rarely dichotomously forked, the apices spirally coiled or hooked; perithecia dorsiventral, generally becoming detached.

Subfamily Phyllactineæ

Mycelium superficial and sending branches through the stomata into the substomatal intercellular spaces, or mycelium intercellular and emerging to the surface to form the perithecia.

Oidiopsis.—Mycelium intercellular; asci several; appendages as in Erysiphe; perithecia dorsiventral; conidiophores branched, emerging through the stomata.

Phyllactinia.—Mycelium with haustoria-bearing branches entering the stomata; asci several; appendages of two kinds: (1) rigid with bulbous base and pointed extremity; and (2) penicillate and transformed into a mucilaginous cap at maturity; perithecia dorsiventral, becoming detached; conidiophores simple, with single conidium.

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POWDERY MILDEW OF APPLE

Podosphæra leucotricha (E. & E.) Salm.

The powdery mildew of the apple is a fungous disease that attacks the 1-year-old shoots, affecting twigs, foliage, blossoms and fruits, disfiguring, stunting, deforming or killing the invaded structures.

History.—This apple disease was first noted in the United States in 1871 by Bessey, who reported it on seedling apples in the nursery at Iowa State College. He referred the fungus to another species of Podosphæra, and it was not until 1888 that Ellis and Everhart recognized the difference and described it as *Sphærotheca leucotricha*. Burrill in 1892 changed the name to *S. mali* (Duby) and it was considered under this name until Salmon in 1900 established the identity of the fungus and referred it definitely to *Podosphæra leucotricha*.

The first extensive experimental work on the control of apple mildew was published by Galloway in 1889, from studies made in the eastern United States, where he found the disease serious on young trees in the nursery. Later he reported the successful use of ammoniacal copper carbonate in controlling the disease. Pammel discussed the occurrence of the disease in Iowa in 1894, and recommended the use of Bordeaux mixture instead of ammoniacal copper carbonate. Brief reports of the occurrence of apple mildew both east and west of the Rocky Mountains were recorded later, but the first extensive consideration of apple mildew as a serious disease was published by Ballard and Volck (1914) from their studies in the Pajaro Valley, California. This was soon followed by the work of Fisher (1918, 1920, 1922), who considered the apple mildew and its control in the arid regions of the Pacific Northwest.

This disease was known only from North America until 1898, when it was reported by Magnus from the Tyrol, but it seems probable that the apple mildew of Germany described by Sorauer in 1889 should be referred to *P. leucotricha*. What was apparently identical with our American apple mildew was studied by Cobb as prevalent in Australia as early as 1892, while Cunningham (1923) has recently given attention to the disease in New Zealand. The apple mildew of England, which occurs in the conidial stage only (*Oidium farinosum*), should probably be referred to *P. leucotricha* also.

Geographic Distribution.—Apple mildew has been reported from many parts of the world, including various European countries, Japan, Australia, New Zealand, the United States and Canada. In the eastern United States it has long been present as a disease of nursery stock, and has been commonly noted on water sprouts of bearing trees and occasionally on the normal twigs of the old trees, but it has not generally been sufficiently troublesome to require attention in commercial orchards. In a few localities in Virginia, West Virginia, Maryland, and other eastern states the disease has been sufficiently severe to demand control measures. In recent years, however, the disease has assumed importance as a disease of bearing orchards west of the Rocky Mountains. While the disease is now found quite generally established in this portion of the United States, there are several important apple sections in which the trouble has developed unusual severity. Special mention should be made of the serious aspects of the disease in the Pajaro Valley of California, where its development has been promoted by the especially favorable climatic conditions,—moderate tem-

peratures with frequent fogs interspersed with clear periods. The drenching of the foliage with fog and dews is common in this valley. Equally severe injury is caused in other but less important apple districts of California. Mildew has caused more or less concern in Utah, Idaho, Oregon and Washington, but it has increased in severity in the central, warm irrigated, valleys of Washington, which include the most important apple districts of the state (Walla Walla, Yakima and Wenatchee). While the disease is very common on the coast, it does not seem to cause so much injury. It seems probable that the absence of drenching rains in the more seriously infected districts is a favoring factor, while the climatic conditions and irrigation practices produce a more susceptible condition of the host.

Symptoms and Effects.—The disease is found on the foliage, twigs, blossoms and fruit of the apple.

Upon the affected leaves the disease is first manifested in small grayish or white, felt-like patches of fungous growth. It usually appears first upon the under side of the leaves, which soon become crinkled and curled. When very young leaves are attacked they have a tendency to increase in length but not in breadth, and may become somewhat folded longitudinally. The fungous patches rapidly enlarge and soon cover the entire leaf. In a very short time the affected areas are covered with masses of powdery spores, which give the disease its name and spread it about during the growing season. The affected foliage is rendered hard and brittle and is frequently killed. In any case it ceases to function normally, and a devitalization of the tree with consequent crop reduction results, the severity of which depends upon the portion of the leaves attacked (Fisher).

The fungus causes the same white powdery appearance on the 1-year-old twigs as on the leaves, but by midsummer (July) the powdery condition begins to disappear and gradually the external whitish growth is transformed into a brown felt-like covering in which numerous minute black fruiting bodies are embedded, giving the affected twigs a speckled appearance. The infected 1-year-old twigs are either stunted or killed back, sometimes completely, sometimes only at the tips, which are especially susceptible to attack.

Blossom infection usually results from the overwintering of the fungus in the dormant blossom buds. In this case the entire blossom cluster and attendant leaves are attacked. The floral parts are shriveled and blighted, so that no fruit is produced. On account of the fact that infected buds do not open and spread mildew spores until some time after healthy buds have unfolded (usually about the time normal blossoms are dropping their petals), there is little danger of general "blight" spreading over the blossoms during the blooming period.

Young fruits, however, are frequently attacked shortly after the blossoming period, and infections may remain established on the apples until the skin hardens in midsummer, after which the fungus generally does not persist upon the fruit. The fungus may become established upon the apples either by spore germination upon the fruit or by spreading down the stem frcm infected twigs. In the former case it is usually the calyx end of the apple that is affected, but in the latter the basin alone may be involved. When very young apples are infected their growth is stunted, but a russeting of the skin always results from the presence

of the fungus. This russeting shows a tracery of fine lines, usually as a network, but sometimes so closely woven that a solid patch appears. The expansion of

FIG. 166.—Shoots of apple heavily infested with powdery mildew (*Podosphæra leucotricha*). (*After Fisher.*)

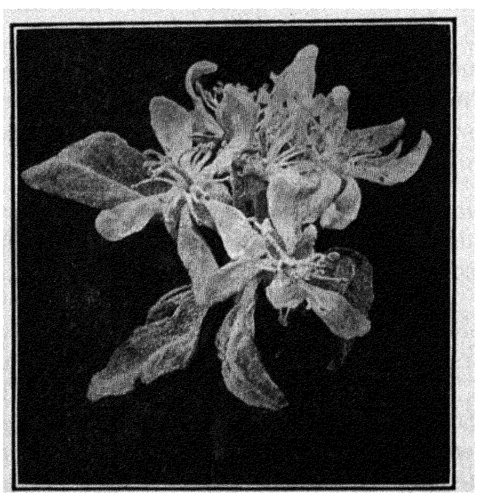

FIG. 167.—Blossoms and young leaves showing severe attack of powdery mildew. (*After Fisher.*)

the growing fruit frequently causes cracks to form in the hardened area and allows the apple to become shriveled (Fisher).

The nature of the disease is such that quantitative determination of the injury or the amount of loss is very difficult. The disease has both a direct and an indirect effect on production. The different types of injury are as follows: (1) the leaves are stunted, deformed or killed, and consequently the tree is robbed of its power of food production, in proportion to the extent of the infection; (2) affected terminals are either stunted or killed back, wholly or in part, the number killed being a partial measure of the injury; (3) the flowers are deformed or blighted so that fruit may fail to set; (4) the blighting of twigs lessens or prevents the formation of blossom buds, which must affect production the following year; and (5) the fruit which does mature may be disfigured by russeting or cracking, so that its market value is considerably reduced. This last effect is not uncommon in Washington, but it is reported as lacking in the Pajaro Valley of California. In general, it can be said that the Pajaro Valley trees in the regions favorable to mildew suffer a gradual decline in vigor and thrifty appearance, and show retarded growth and lessened productiveness unless careful control measures are practiced. In recent years the disease has been very severe in Austria and Czechoslovakia. Baudys has reported the production of witches' brooms in some varieties.

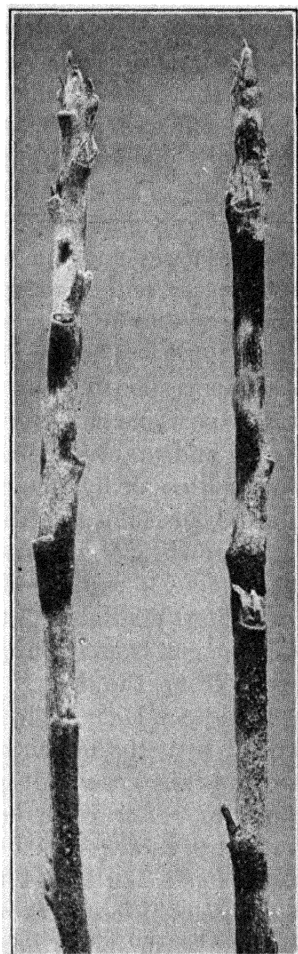

FIG. 168.—Apple twigs in dormant condition, showing a heavy coating of powdery mildew. The perithecia are present in large numbers in the dark patches. (*After Fisher.*)

Etiology.—Apple powdery mildew is caused by an obligate fungous parasite, *Podosphæra leucotricha* (E. & E.) Salm. This fungus, like all other powdery mildews, is directly and absolutely dependent upon its host, so there never has been any doubt as to its causal relation to the disease. Another closely related mildew, *P. oxyacanthæ* (DC.) De By., is sometimes found on the apple in the eastern United States, but it has never been reported as serious.

The pathogene is an external parasite and spreads its delicate, cobweb-like filaments or hyphæ (mycelium) over the surface of the affected parts —leaves, flowers, young fruits and 1-year-old stems. This fungous body absorbs its nourishment from the underlying cells by means of special sucking organs which penetrate the cell cavities. The fungous body pro-

duces two kinds of spores or reproductive bodies—conidia or summer spores, and ascospores or overwintering spores.

As soon as the hyphæ have established themselves on any parts, the prostrate vegetative hyphæ give rise to numerous erect branches which form chains of specialized barrel-shaped cells or conidia. These conidia soon begin to break away at the ends of the chains and, dropping down between the erect hyphæ, produce the characteristic powdery appearance of the affected parts, which has suggested the common name of the parasite. Each conidiophore, or conidia-bearing branch, has an unlimited power of spore production, so that enormous numbers of conidia may be produced on a single leaf. A conidium can germinate at once, and produce a new fungous body if it can find a new host surface on which to develop. Contact with the host surface seems to be very important, since germination is poor in either water or nutritive solutions (Woodward, 1927). The conidia are carried by the wind or by other agents, and are thus responsible for the rapid local spread of the mildew during the earlier portions of the growing period.

By midsummer the mycelium on the diseased twigs has changed from the colorless or hyaline condition to brown, and as the result of a sexual process, special spore fruits, the *perithecia*, appear in the form of minute, dark-brown, globular bodies, barely visible to the naked eye (75 to 96μ in diameter), embedded in the external felt-like growth. Each spore fruit shows a number (3 to 11, usually 3 to 5) of long, rigid outgrowths, or appendages, from the upper side and some short, flexuous hyphæ (appendages) from the lower surface (sometimes nearly obsolete). Young apical appendages are hyaline, thin-walled and septate, but become thick-walled with the lumen more or less obliterated with age, and are dark brown in the lower half but paler towards the tip, which is undivided and blunt or rarely once or twice dichotomously divided. The essential structure, however, is a single, oblong or subglobose, eight-spored sac or ascus (55 to 70 by 44 to 50μ) within the perithecium. The hyaline, single-celled ascospores reach maturity in the spring of the year and during the warm spring rains the perithecia rupture and the spore sacs are forcibly expelled and then explode with the expulsion of the ascospores, or the protruding spore sacs may explode without being expelled (Woodward, 1927). Thus the ascospores are forcibly expelled, and may be carried away by air currents, and fall on young foliage, when new infections may be established. In the apple the perithecia appear to be confined very largely to the twigs but may occur on the petioles, midrib and larger veins. On the pear they are rare on the twigs, but may sometimes be produced in abundance on the fruits (Fisher, 1922).

From the above account it might be inferred that the ascospores are the only means of carrying the fungus over the winter, but it was pointed out by Galloway in 1889 that they were of little importance, and that the

fungus overwinters in the form of a dormant myeelium in the leaf and flower buds. This opinion has been corroborated by the work of Ballard for the Pajaro Valley, California, by Fisher for the Wenatchee district of Washington, and by Woodward for England. According to Ballard and Volck:

> The mildew growing over the surface of the shoots has an excellent opportunity to work its way in between the bud scales and penetrate both the lateral and terminal buds. This actually happens, and within these buds the mildew passes the winter in a dormant condition. The following spring, as the infected buds begin to open, the mildew commences to grow and keeps pace with the development of new leaves and twigs. Hence infected shoots appear all over the trees as soon as they leaf out in the spring.

The unimportance of the ascospores in the immediate spread of the fungus is emphasized by the rarity of the spore fruits in certain sections,

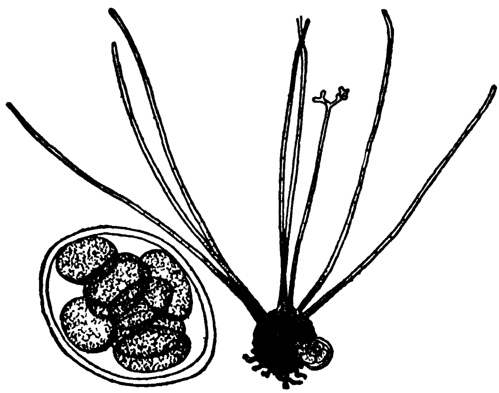

FIG. 169.—Perithecium of *Podosphæra leucotricha* with ascus being liberated; also a free ascus much enlarged. (*After Fisher, U. S. Dept. Agr. Bul.* **712.**)

especially England and the eastern United States. Fisher has pointed out that mildew is in evidence in the Wenatchee district before the ascospores are expelled, and concludes that the ascospores play little if any part in spreading the disease. It seems especially significant, however, that a copious production of ascospores is characteristic of those regions in which mildew is severe. The short-lived nature of the conidia or summer spores would suggest that they are responsible for the immediate local spread of the disease, while the more resistant ascospores may be responsible for a wider dissemination and rejuvenation of vigor or virulence.

Host Relations and Varietal Resistance.—While this mildew is of most importance as an apple disease, it also affects the pear, quince, cherry, plum, hawthorn and june berry. It is sometimes the cause of serious injury in pear orchards, especially in those regions of severe infestation of apples, like the Yakima and Wenatchee valleys. As observed by the

writer in the Yakima Valley, and by Fisher in the Wenatchee district (1922), twig and foliage infection is not general, but the fruit may be very generally attacked. D'Anjou and Louise Bonne are recorded as susceptible, Bartlett moderately susceptible and Flemish Beauty and Winter Nelis markedly resistant. The effect of the disease on the pear is

> . . . the production of a black or russeted disfiguration, and in some cases a distortion of the shape somewhat like early scab infection. There is also evidence that the disease caused an abnormal drop of fruit, especially on D'Anjou (Fisher, 1922).

So far as known, no varieties of apples are immune from powdery mildew, but varying degrees of resistance are shown. The following varieties have been listed with reference to their susceptibility:

Susceptible	Location	Resistant	Location
Black Ben Davis........	Wenatchee Valley	Langford	Pajaro Valley
Esopus (Spitzenburg)...	Pajaro Valley Wenatchee Valley	Red Astrachan	Pajaro Valley
Fameuse (Snow)........	Wenatchee Valley	Rhode Island Greening	Pajaro Valley
Grimes Golden...... ...	Wenatchee Valley	White Permain	Pajaro Valley Wenatchee Valley
Gravenstein............	Pajaro Valley	Red Permain	Pajaro Valley
Jonathan..............	Wenatchee Valley	Winesap	Wenatchee Valley
Missouri...............	Pajaro Valley		
Smiths' Cider..........	Pajaro Valley		
Stayman...............	Wenatchee Valley		
Yellow Bellflower.......	Pajaro Valley		
Yellow Newtown.......	Wenatchee Valley Pajaro Valley		

In England, 10 varieties are classed as medium in susceptibility, 8 as very susceptible, while 2, Norfolk Beauty and Worcester Pearmain, are classed as highly resistant (Woodward, 1927).

Control.—In the early experiments on the control of apple powdery mildew in the eastern United States both Bordeaux and ammoniacal copper carbonate were recommended. The development of the disease in severe form in the regions west of the Rocky Mountains presented new control problems, peculiar to the different climatic conditions. Three distinct lines of procedure have received emphasis: (1) pruning; (2) cultural or other practices to produce vigorous early growth; and (3) spraying. In the regions of severe infestation no single practice will give adequate control. In well-cared-for orchards, in which it is possible to use lime sulphur for scab, mildew causes little or no additional concern.

1. *Pruning.*—Winter pruning for the removal of mildewed shoots which carry the fungus over the winter should be practiced. During the

dormant season prominent infections may be detected by the gray or silvery appearance of the twigs. Careful attention to the elimination of these mildewed shoots will lessen the amount of the disease that will appear when growth starts in the spring and consequently will retard the spread of the disease. Careful attention to pruning is valuable for other reasons. Winter pruning improves the growth and foliage conditions during the following season and consequently is of advantage in combating the mildew. In this operation interlacing branches should be removed and the long, spindling branches cut back. In severely mildewed orchards it will probably be advisable to prune much more heavily than under perfectly normal conditions.

2. *Cultural or Other Practices.*—Careful attention to cultivation, cover crops or irrigation methods suited to the locality will help by keeping the trees in the best possible vigor. Puny, slow-growing trees are more susceptible to mildew than those with rapid vigorous growth. Spraying with crude-oil emulsions, as commonly practiced in the Pajaro Valley for scale-insect control, is reported to stimulate a vigorous early growth of foliage the following spring, and consequently to be of value in mildew control. For soils weak in nitrogen the use of sodium nitrate as a fertilizer may be of material aid, but overfertilization should be guarded against, as an excess of nitrogen is favorable to mildew.

3. *Spraying.*—The first two control practices are at best only supplements to spraying, which must be practiced in orchards of the regions favorable to mildew. The times for application of the spray mixtures are as follows: (1) just before the blossoms open (pink spray); (2) just as the last petals are falling (calyx spray); (3) about 2 weeks after the calyx spray; (4) about 4 weeks after the calyx spray. In cases of severe infection it may be necessary to apply later sprays which may be put on at intervals of about 3 weeks until the latter part of August. These spraying dates are the same as those recommended for scab (see p. 625), but scab is either absent or present in only small amount in most regions seriously affected by mildew. It should be noted that the first spraying was omitted from the recommendations for the Pajaro Valley, California, but it seems probable that it could be used to good advantage there also.

The materials recommended for the different applications are as follows: For "pink spray," "calyx spray," and the third application use either lime sulphur, 1–50, or iron sulphide; for later applications use lime sulphur, 1–50, or iron sulphide in regions where there is no danger of burning or in case of light crops or severe infections in regions where burning is likely, or where sulphur sprays cause burning use ammoniacal copper carbonate (3–5–50 formula), neutral Bordeaux (4–4 $\pm$ 50 formula) or Burgundy mixture with lime (4–5–2–50 formula).

The climatic peculiarities of the Pajaro Valley, which caused much injury when either Bordeaux or lime sulphur was used, led to the substitu-

tion of the iron sulphide mixture by Ballard and Volck. While this was satisfactory for that region, its adoption for the hot irrigated valleys of central Washington led to disastrous results.

In the Yakima Valley and in the Wenatchee Valley also, both the iron sulphide, lime sulphur, and other sulphur fungicides have caused much burning of exposed fruit (sulphur sun scald) when used after about June 1, when high temperatures (above 90°F. in the shade) and burning sunshine are common. The copper sprays have been introduced to overcome this difficulty. In general, they are less efficient in mildew control than the sulphur sprays, and copper carbonate is much more expensive than the sulphur mixtures. It has also been pointed out that it is unsafe to follow a lime-sulphur spray with summer oil because of the resultant defoliation and fruit drop (Fisher, 1928).

Various proprietary preparations of sulphur consisting of very finely divided particles are also effective in the control of powdery mildew, especially for protective sprays, but lime sulphur is more effective when it is necessary to fight well-established cases of active mildew. Atomic sulphur, 10 pounds to 100 gallons of water, has been recommended by Cunningham (1923) as the most efficient fungicide for New Zealand. Dusting with sulphur gave satisfactory control in nursery stock (Stewart, 1915), and Fisher (1928) reports orchard control with a new type of dusting sulphur that is activated by the addition of potassium permanganate. "Pomastin," a preparation of coal-tar components, gave satisfactory field control (Schubert and Richter, 1926).

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IMPORTANT DISEASES CAUSED BY POWDERY MILDEWS AND ALLIES

I. ASPERGILLACEÆ

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II. PLECTODISCELLACEÆ

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III. Perisporiaceæ

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CHAPTER XXII

DISEASES DUE TO THE SPHERE FUNGI AND ALLIES

PYRENOMYCETES

The fungi belonging to this group have no distinctive feature of either mycelium or conidial fruits. The latter generally constitute a very prominent stage in the life history of these pathogenes, while the ascigerous stage, which is frequently formed independent of the host or as a saprophyte on dead parts, is in many cases much less conspicuous. Various types of conidial fruits may be developed and when these alone are known the fungi forming them are referred temporarily to the proper groups of the *Fungi Imperfecti*.

The Ascigerous Fruits.—The distinctive feature of the sphere fungi and allies is the ascigerous fruit, which is typically an ostiolate *perithecium*. This is a globular or sac-like structure enclosing the asci, which generally originate in a group from the basal portion of the cavity. Paraphyses or sterile filaments may be present or lacking. The perithecia may be simply sac-like cavities immersed in an aggregate of fungous tissue, a stroma, or they may be provided with definite walls. In the latter case the perithecia may be single and embedded in the substratum, seated upon it, or they may be grouped in stromata that are either embedded in the substratum or are superficial. The sphere fungi and allies as here considered constitute a large part of the group of *Pyrenomycetes*, while the cup fungi and allies are the forms included under the *Discomycetes*.

The asci are typically eight-spored and the ascospores may be hyaline or dark, show great diversity of form and vary from continuous to many-septate. As in the cup fungi, the ascospores are forcibly expelled at maturity, but only a single ascus explodes at once. In a typical case an ascus elongates until the tip protrudes through the ostiole, and when the hydrostatic pressure within the ascus becomes sufficient the explosion occurs and the spores are shot forcibly upward into the air. Following the collapse of the old ascus wall another ascus may elongate to take its place and soon another spore load is shot into the air. Spore expulsion continues during favorable conditions of temperature and moisture until the asci have all exploded. Perithecia are thus repeating spore guns which are sending ascospores into the air to be carried away by air currents. In a few cases the asci do not explode but the ascospores are set free by the gelatinization of the asci.

Classification.—The following is a tabulation of the orders and families with the important genera furnishing plant pathogenes.

I. Hypocreales

The perithecia are soft leathery, fleshy or membranous and are *never black* but show various other colors, such as buff, yellow, brown, red or purple. The mycelium is also light or bright colored but never black. The perithecia are globular or flask-shaped, free on the substratum, embedded in a superficial mycelial weft or subiculum or seated on or sunken in a stroma. In general, the ostiolate perithecia have a thick wall of more or less pseudoparenchymatous character, but in Claviceps the walls of the perithecia are not differentiated from the stroma.

1. Hypocreaceæ.—A single family with the characteristics of the order.

Nectria.—Perithecia soft membranous or fleshy, yellow, red or brown, single or grouped on the substratum, on a subiculum or in or on a fleshy stroma. Spores two-celled, ellipsoid, blunt or pointed, generally hyaline, but rarely reddish. Conidial stages prominent and various.

Calonectria.—Perithecia red or yellow, free or seated in a white cottony radiate subiculum. Spores elongated, hyaline, four-celled.

Gibberella.—Perithecia transparent blue or violet, free or seated on an effused or fleshy stroma, and scattered or grouped. Spores ellipsoid or fusoid, four-celled, hyaline. Conidial stage a Fusarium.

Claviceps.—Develops in the ovaries of grasses or sedges and forms horny sclerotia which replace the seed. Overwintered sclerotia produce a number of cylindrical stalks each bearing a globular head (stroma or receptacle) in the periphery of which the asci are borne in perithecial cavities without differentiated walls. Asci eight-spored, ascospores continuous, fascicled, needle-shaped and hyaline. A conidial or Sphacelia stage is formed during the early period of development of the sclerotia.

Ustilaginoidea.—Conidia-bearing sclerotia replace grain of host; conidia continuous, globose, greenish, echinulate; perithecia in a stroma as in Claviceps.

Epichlœ.—Stromata smooth, fleshy membranous, surrounding the culms and leaf sheaths of various grasses, white at first, becoming reddish brown. Perithecia immersed, spores filamentous, breaking up within the asci.

Cordyceps.—Develops in the bodies of adult or larval stages of certain insects, a few in subterranean fungi, transforming them into mummies or sclerotia, from which stalked stromata are developed. Perithecia embedded in the enlarged terminal portions. Ascospores filamentous, breaking up within the asci.

II. Dothidiales

Mycelium forming a sclerotia-like stroma, generally within the substratum, but becoming free by the rupture of overlying host tissue. *Stromata black* superficially but pale or light colored in the interior (stromata are sometimes superficial from the first or remain permanently immersed). The perithecia are simply ascus-containing locules in the stromata and are without differentiated walls.

1. Dothidiaceæ.—Characters as indicated for the order.

Phyllachora.—Stromata in the mesophyll of leaves, remaining covered by the epidermis. Ascospores continuous, hyaline.

Plowrightia.—For the most part in branches of trees or shrubs and forming a compact superficial stroma on which the perithecia are borne. Ascospores ovate, two-celled and hyaline.

Dothidella.—Similar to Phyllachora, but ascospores hyaline, unequally two-celled.

III. Sphæriales

The Sphæriales, or the sphere fungi, are characterized by the formation of typically ostiolate, mostly globular perithecia with well-developed walls, which constitute a distinctive character as contrasted with the Hypocreales and the Dothidiales. The perithecia are carbonous, soft leathery or membranous, brittle, tough or delicate, single or grouped and may be immersed, erumpent or superficial on the substratum or in connection with either a subiculum or a well-developed stroma. The ostiole of the perithecium is sometimes only a circular opening in the free end but more frequently it is at the tip of a short papilla, an elongated beak or a long, slender neck, many times the length of the body of the perithecium proper. The various forms of ostioles may be found in both free and stromatic species. The spores are either hyaline or dark and great diversity is shown in form and septation, varying from globular to acicular and from continuous to septate forms, either linear aggregates or muriform masses. The light or dark (mostly the latter) mycelium is either in or on the substratum and may be filamentous or compacted into stroma-like aggregates.

The Sphæriales consist of numerous families which differ from each other by only slight characters. The number of genera and species is very large, and while the number of parasitic species is small as contrasted with the saprophytic forms, the group furnishes a considerable number of very important plant pathogenes. The saprophytic forms may occur on various substrata, but they are especially abundant on decaying herbaceous stems, woody twigs or leaves. Of the parasitic species, the great majority have retained the saprophytic habit for the perithecial stage, while a conidial stage develops on the living parts of the host. The

perithecial stages may reach maturity only after the old dead host tissues (stems, twigs, cankers, fruits or dead leaves) have been subjected to winter temperatures, and for this reason the ascospores have sometimes been designated as the "winter spores," while the conidia have been called the "summer spores." This condition does not always prevail, but in some forms ascospores are matured whenever conditions for growth are afforded, while conidia may be developed even during the winter period when the temperatures are moderate.

The most important genera of the various families furnishing plant pathogenes will be given brief characterization in the following key, based principally upon spore characters

I. Spores one-celled, hyaline:
  A. Perithecia clothed with hairs.......... *Trichosphæria*
  AA. Perithecia smooth:
    B. Stroma lacking:
      Perithecia long-beaked and single.......... *Ceratostomella*
      Perithecia long-beaked and in a valsoid cluster.......... *Calosphæria*
      Perithecia papillate or apapillate
        Paraphyses present.......... *Physalospora*
        Paraphyses lacking (see also Guignardia).......... *Ascospora*
    BB. Stroma valsoid:
      Spores slender, curved.......... *Valsa*
      Spores elongated, cylindrical.......... *Cryptosporella*
      Spores fusoid.......... *Botryosphæria*
    BBB. Stroma not valsoid, perithecia rostrate.......... *Glomerella*

II. Spores one-celled, dark:
  A. Perithecia superficial or sunken in the substratum, separate:
    Apapillate.......... *Rosellinia*
    Spiny rostrate.......... *Acanthorhynchus*
  AA. Perithecia in a stroma:
    Stroma disk-like.......... *Numullaria*
    Stroma pulvinate.......... *Hypoxylon*
    Stroma club-shaped or branched.......... *Xylaria*

III. Spores two-celled, hyaline:
  A. Stroma dark, diatrypoid or valsoid.......... *Diaporthe*
  AA. Stroma orange to brown, valsoid.......... *Endothia*
  AAA. Stroma lacking:
    B. Perithecia smooth, immersed or protruding:
      C. Asci thickened at the end and provided with a terminal canal *Gnomonia*
      CC. Asci not thickened or without a terminal canal.
        Spore cells very unequal, small lower cell cut off shortly before maturity.......... *Guignardia*
        Spore cells equal or nearly so.......... { *Mycosphærella* / *Didymellina* }
    BB. Perithecia setose at apex, immersed and later protruding or superficial.......... *Venturia*

IV. Spores two-celled, smoky or dark:
  A. Perithecia many paraphysate, carbonous, superficial.......... *Neopeckia*

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BLACK KNOT

Plowrightia morbosa (Schw.) Sacc.

This characteristic disease of plums and cherries is readily recognized by the black, elongated galls which appear on twigs and branches. It has been called plum wart, but black knot is the name most generally used.

History and Geographic Distribution.—Black knot was noted in Massachusetts in destructive form over a hundred years ago (1811), and since that time has been treated in popular and scientific literature. During the earlier years of its consideration two different ideas prevailed as to the origin of the knots: (1) that they were due to a diseased condition of the sap; (2) that they were the result of insect stings. The first intimation that the knots were of fungous origin was in 1843, when it was shown that the curculio merely lays its eggs in young juicy tissues that are already diseased. The ascigerous stage of the pathogene had previously been described by Schweinitz (1821), but he appeared to regard the combined action of a gall fly and the fungus as the inciting cause of the hypertrophies. The idea of the insect relation was gradually abandoned as more attention was given to the fungus. The first complete account of the structure of the fungus was published by Peck (1872), and a few years later (1876) Farlow presented a fuller account of the life history of the fungus which left little doubt as to its causal relation to the knots. Later studies, by Crozier (1885), Scribner (1890), Humphrey (1891, 1893), Halsted (1891), Beach (1892) and Lodeman (1894) added somewhat to our knowledge of the disease and considered its prevention or control. During recent years but little detailed study has been devoted to the disease.

Black knot was especially abundant in the New England states when attention was first directed to it, and it is believed to be of American origin. It probably first affected wild species and spread from these to the cultivated varieties of plums and cherries. Whether the disease originated on the Atlantic seaboard and gradually spread westward is uncertain. There are some indications that it was present in western localities long before attention was directed to it. By 1879 there were reports that the disease had appeared for the first time in the vicinity of Cincinnati. While it spread westward it never became so common or so severe in the Middle West as on the Atlantic seaboard. The disease is rare in the regions west of the Rocky Mountains, except on wild species. It appears very abundantly on the native chokecherry (*Prunus demissa*) in Washington and adjacent territory, but does not attack cultivated species even when they are growing in close proximity to thickets of the wild host. During an experience of 17 years in Washington not a single authentic case of the occurrence of the disease on cultivated species has been brought to our attention. The disease is not known to occur outside of America.

Symptoms and Effects.—The black knot is first in evidence as a slight swelling of a twig or branch, either adjacent to an old knot or separate from it. While this swelling may sometimes be observed in the fall, it becomes conspicuous in the spring after growth starts and the bark soon ruptures and a straw-colored or light yellowish-brown, granular growth fills the crevices. As the season progresses the overgrowth becomes more pronounced and somewhat darker. In the late spring or early summer the smooth surface of these excrescences shows a pale-greenish tinge at places and soon the entire surface becomes an olive green and appears covered with a velvet-like pile. This velvety surface soon disappears, the knot becomes darker and by late fall it has become perfectly black.

The knots vary in location, shape and size. They may appear on young twigs or on older branches up to about 2 inches in diameter, and may originate at any of the following places: (1) crotches of limbs; (2) at

the union of the growth of consecutive seasons; (3) on fruit spurs; (4) at the tips of twigs; and (5) in the axils of leaves or about an axillary bud. Some of the knots may be short ($\frac{1}{4}$ to $\frac{1}{2}$ inch), but they generally extend for some distance along a branch (several inches to a foot), and are more or less fusiform, although they may sometimes be nearly uniform in diameter for the entire length. Adjacent knots may sometimes fuse to form much more extended excrescences. Their diameter depends largely on the size of the structure on which they form, so they may be only a fraction of an inch or 1 or 2 inches in diameter. It is typical for the knot

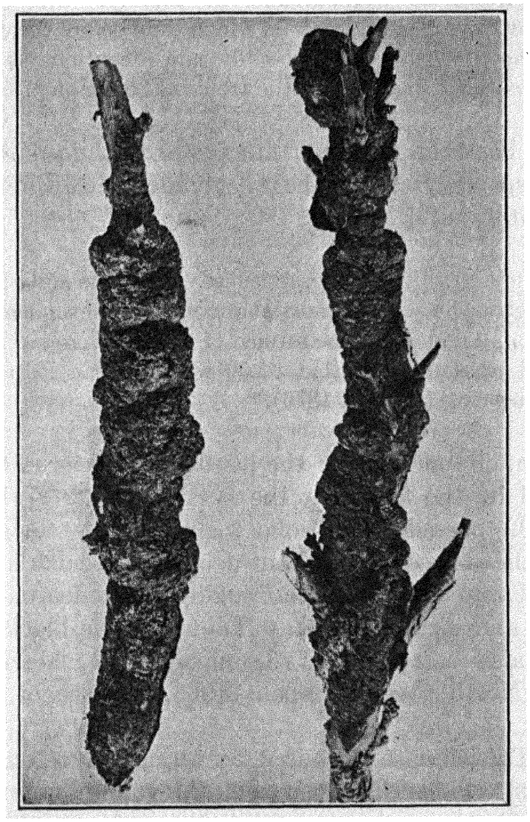

FIG. 174.—Black knot on Japanese plum.

to be confined to one side of the twig or branch, yet at times the branch is completely encircled. The affected structures may be straight or curved or even thrown into more or less of an irregular spiral. If knots are formed at the forking of branches, they generally extend along both branches and also down on the main axis.

In a tree in which the disease has been allowed to run its course undisturbed for a number of years the following should be found if the tree is examined in June: (1) young excrescences developed during the spring

growth; (2) old knots that matured during the early spring; and (3) old knots developed during previous seasons, which are likely to be more or less eaten by insects and are frequently infested with saprophytic fungi. Some of the new knots may be extensions of old ones rather than representing new infections. The character of the injury may be expressed as follows:

> Small twigs are frequently killed outright, for in them the disease soon cuts off all communication between the parts above and those below the affected portion. Large limbs do not succumb so rapidly. The disease gradually extends from year to year and it may be a long time before the death of the parts beyond takes place, for such, with possibly rare exceptions, is the result which eventually follows the appearance of the trouble (Lodeman, 1894).

Black knot has been an important factor in the production of plums and cherries, especially east of the Alleghanies and from North Carolina to southern Ontario and Maine.

> In some parts of New England, particularly in Maine and along the seacoast, the raising of cherries has been almost abandoned in consequence of the ravages of the black knot. An idea may be formed of the small crop of plums now raised in New England from the fact that $2.50 was given in Boston for a peck of Damsons for preserving (Farlow, 1876).

The economic importance of the disease at a later date in New York may be judged by the fact that the Western New York Horticultural Society in 1892 passed resolutions including the "demand that the Legislature of the state shall, without delay, enact such a law as shall, in its enforcement and execution, thoroughly and effectually exterminate that infectious and incurable disease known as the black knot" (Beach, 1892). It is also stated that the growing of cherries was nearly abandoned in North Carolina about 1906, because of the ravages of black knot.

Etiology.—This disease is caused by an ascomycetous fungus which produces at least one *conidial stage* when the young knots appear velvety and olive green, and an *ascigerous stage* in the surface of the black matured knots. The pathogene was first named *Sphæria morbosa* by Schweinitz in 1821, and some other generic names were proposed later, but none came into general use until Saccardo assigned the fungus to the Dothidiales and called it *Plowrightia morbosa* (Schw.) Sacc., by which name it has been known until recent years. Some differences of opinion have arisen as to its real relationship, as witnessed by the recent assignment to a newly recognized order, the Pseudosphæriales (Theissen and Sydow, 1918), in which it appears as *Dibotryon morbosum* (Schw.) T. & S. More recently it has been assigned to the Melogrammataceæ and the genus

Botryosphæria (Sorauer, fourth edition, **2**: 328–330, 1921). The causal relation is based largely on the presence of mycelium in the young knots and on the perennial nature of the infections. Aside from the early statement of Farlow (1876) that "we have made direct experiments to show that the spores of the knot on the chokecherry will germinate and produce the knot in healthy plum trees," successful inoculations have not

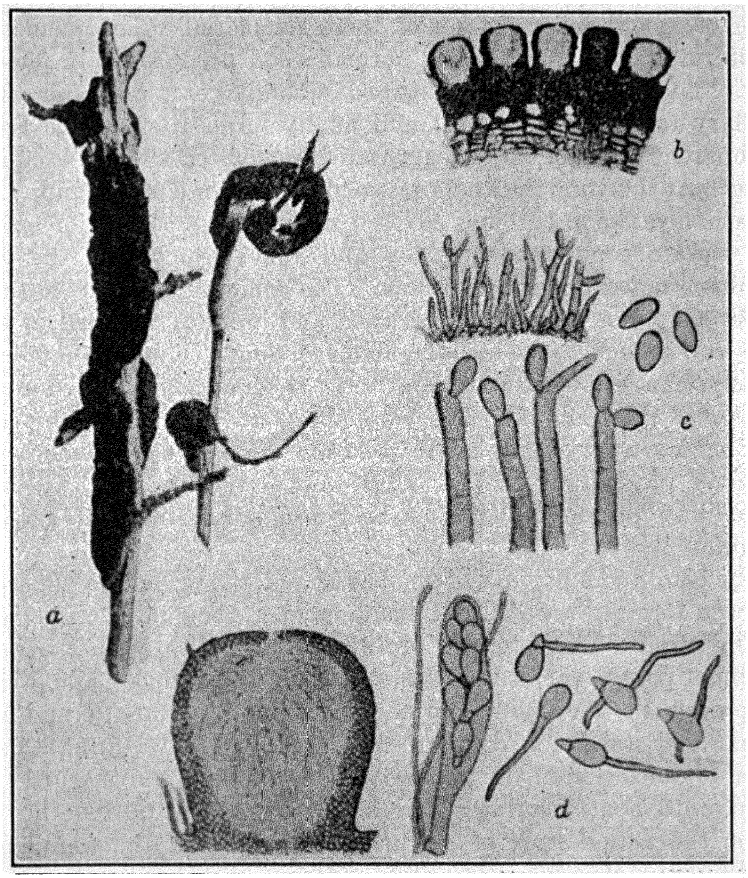

FIG. 175.—*Plowrightia morbosa.* *a*, habit sketch; *b*, section of stroma and perithecia; *c*, conidiophores from the surface of a young knot with several conidiophores and spores more highly magnified; *d*, section of a single perithecium, an ascus and paraphyses, and several ascospores germinating. (*After Longyear.*)

been reported. With our present information there are several blanks in the life history of the pathogene, the part played by the different spore forms, and the exact time and manner of infection being based on observational data.

Knots may be *primary*, that is, the direct result of infection from spores, or *secondary*, due to the invasion of new portions by the mycelium that is already present in the tissues. Mycelium comes to be present in

the cambium, and when growth starts, the cells are stimulated to an abnormal activity and both wood and bark are affected. The mature gall is a complex of disarranged cells, the wood and bark undergoing about proportional increase in thickness. According to Stewart (1914), the cambium retains its normal position between xylem and phloem, except opposite the broad medullary rays, where it may be broken up and pushed out into the cortex, and then give rise to isolated xylem elements or groups of xylem cells. Many of these misplaced xylem elements are scalariform tracheids, while the normal wood produces only pitted elements. The mycelium grows outward in bundles or strands following the medullary rays (Farlow, 1876) and finally forms a stromatic layer, or pseudoparenchyma, of closely aggregated fungous cells which occupies the surface. At this time the knots are solid but rather fleshy or pulpy. The stromatic layer soon becomes covered with a dense coating of brownish, erect, septate conidiophores (May and June) which give the velvety appearance noted under Symptoms. The conidiophores are 40 to 60 by 4 to 5μ, simple or sometimes branched and produce terminal or lateral, brown, continuous, ovate conidia, about 6μ long. The conidiophores are generally somewhat flexuous, and may become geniculate due to the extension of the terminal cell beyond the point of origin of the conidia. The conidia are very easily separated from the points of attachment and a copious production follows until about midsummer. From their position and powdery character they are admirably suited to wind dissemination.

Even before conidial production has ceased, the primordia of perithecia have been forming beneath the conidiophores, their position being indicated by minute hemispherical protuberances, visible with a hand lens. As summer passes, the conidiophores dry up, the surface papillæ become more prominent, the knots become harder and more brittle and assume the characteristic black color of the mature galls. During this time internal organization of the perithecia is proceeding, but maturity is not reached until nearly spring. The asci grow slowly during the winter months, the actual time of maturity varying with the temperatures. Farlow found mature ascospores in abundance in February, but observations as to the exact time when they are discharged seem to be lacking. Each perithecium seems to be a single, ostiolate cavity within a stroma-like aggregate of fungous tissue which makes up the body without any well-defined wall. Each locule or perithecial cavity is filled with asci and sterile, filiform, non-septate filaments with slightly enlarged tips. The asci are about 120μ in length and contain eight, hyaline, one-septate spores, the lower cell being uniformly shorter and narrower than the terminal cell. The ascospores are obliquely uniseriate or irregular in arrangement and measure 8 to 10 by 16 to 20μ. Under suitable conditions of moisture and temperature the mature ascospores are able to

germinate in 1 to 5 days. One or more hyphæ may grow out from the large cell and none or only one from the small cell.

Farlow (1876) described three types of pycnidia which he found mingled with perithecia: (1) one with yellowish septate spores, since named *Hendersonula morbosa* by Saccardo; (2) a Phoma-like form with hyaline spores liberated in tendrils; and (3) a Phomopsis-like type, which he called spermogonia. Humphrey (1891) later described a pycnid with single-celled brown spores which he thought belonged to the black-knot fungus but he was unable to reproduce the disease by inoculations made with these spores from pure cultures. Some or all of these pycnidial forms are either saprophytic on the old knots or possibly parasitic on the black-knot stromata.

It is the prevailing opinion that infections, whether originating from conidiospores or from ascospores, take place soon after the dissemination of these reproductive bodies, since they are capable of immediate germination. Lodeman (1894) showed that in certain cases the external cork layer may be interrupted by a fissure leading into the cortex in the axil of a branch, and expresses the opinion that infection takes place through such fissures or at some other points where the external cork layer is poorly developed. However infection takes place, the formation of a knot apparently does not begin until the mycelium of the pathogene reaches the cambium. In most cases it seems that cambial activity has subsided or is at a low ebb by the time the mycelium reaches the cambium, and consequently only a few of the knots become evident in the fall following the time of infection. These, however, grow rapidly in the spring and produce the conidial stage and later the perithecia which are mature and ready for ascospore dissemination in the spring of the next season.

Host Relations.—The black-knot pathogene attacks various species of plums and cherries. It is especially in evidence on many wild species, but is sometimes noted on one species when an adjacent species, perhaps, with interlocking limbs remains free, and wild and cultivated species may be in proximity without both becoming infected. Stewart (1914) records that "the chokecherry and wild plum (*P. americana*) are infected in the vicinity of Madison, Wis.," and further states that the wild black cherry sometimes growing in thickets of badly infested chokecherries remains free from knots. Reference may be made again to the conditions in the Pacific Northwest, where the native chokecherry (*P. demissa*) is very generally affected, while cultivated plums and cherries remain free from the disease. The probable explanation for this behavior is the existence of biological species or strains that have become so adapted to certain hosts that they are unable to infect others. Farlow (1876) called attention to the fact that no morphological difference could be detected between the fungus on the cherry and the one on the plum.

The opinion has been expressed that there is no variety of cultivated plum that is not subject to the disease. Farlow (1876) believed that the disease spread from either the bird cherry (*P. pennsylvanica*) or the chokecherry (*P. virginiana*) to the cultivated plums, since these species are common in the vicinity of Boston, while the wild plum is very rare there or does not occur at all.

The Trifloras are said to be affected less than any other group of plums, and the Institias rank next in immunity, although the Damson is said to be very susceptible. On the other hand, the Domesticas are susceptible, except possibly the Middleburg and Palatine, which are relatively free from black knot (Hesler and Whetzel, 1917).

In general, cherries are reported to suffer less than plums, although their behavior seems to vary with the environment. In Ontario, cherries are said to suffer more than plums. Sour cherries, especially the English Morello, are very susceptible to black knot, while the Mazzard comes next (Farlow, 1876). Sweet cherries are so resistant that they are seldom infected, although a few cases have been recorded.

Control.—The practices which have been recommended for the elimination or control of the disease are: (1) the cutting out of groups or thickets of wild cherries or plums adjacent to cultivated orchards; (2) surgical treatment of affected cultivated trees; (3) spraying to prevent new infections. The need of removing the wild hosts will vary with the environment, since in some localities they are not a menace to the cultivated species. Whenever it is known that the disease is passing from the wild hosts to the cultivated species, other attempts at control will be partially nullified if infected wild hosts are allowed to remain nearby.

In the treatment of cultivated trees several features should be kept in mind: (1) that the fungus is perennial in affected branches and extends beyond the external evidences of the knots; (2) that conidiospores are matured when the young knots show the olive-green velvety coating; (3) the ascospores are matured early in the spring on old knots; (4) that spores are said to ripen on the brush pile. The objective in surgical treatment is, first, to remove all of the perennial mycelium so that secondary knots cannot be formed from its extension, and, second, to remove and destroy the knots previous to the liberation of spores in order to prevent new infections. If pruning is started in time, the sacrifice of the affected parts will not seriously thin out the branches. The cuts should be made several inches below the external evidence of the knots, and the prunings should be collected and burned. The knots should be removed when they show yellowish brown in the spring, that is, before the conidia have matured. A second inspection should be made in the fall and any knots that have been overlooked should be cut out, but special emphasis

is to be placed on thorough work in the spring. If the trees are gone over carefully twice previous to the formation of the conidia, few knots should escape. In regions in which outside sources of infection can be largely eliminated, pruning alone should serve to hold the disease in check or to eradicate it. The neglected orchard is a menace, but in many states the painstaking orchardist is protected by legislation for the suppression of the black knot, and inspectors should have no hesitation in enforcing such laws as do exist.

In environments in which surgical treatment is not effective the grower is left the alternative of spraying or of giving up the growth of susceptible trees. The latter has happened in a number of cases, but it has been shown that spraying will very greatly reduce infections. In no case should main reliance be placed on spraying, since at its best it must be considered supplemental to the surgical treatment. The recommendations for spraying are based largely on the work of Lodeman (1894), who suggested the following applications of Bordeaux: (1) during late March or early April; (2) when buds are beginning to swell; (3) during the latter part of May, or when the new knots begin to show the velvety coating; (4) two or three subsequent applications at suitable intervals. While this treatment did not give perfect control, the number of new knots in test plots was reduced from 2000 to 165. It should be realized that these recommendations rest on very meager experimental results, and that carefully controlled tests are needed. It would seem that more certain knowledge as to the time when infections occur, coupled with the use of stickers as employed in modern spraying, should increase the effectiveness of the applications and perhaps make possible a reduction in their number.

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APPLE SCAB

Venturia inæqualis (Cke.) Wint.

The disease of the apple known as scab manifests itself upon either leaves, blossoms or fruits and sometimes appears on the young or 1-year-old twigs. It causes a spotting, discoloration or distortion of both fruit and foliage, but it is never directly responsible for a rotting of the fruit. It is the fruit attacks which cause the serious and frequently enormous losses.

The disease is also referred to as apple scab, scurf, black spot, black-spot fungus, Tasmanian black spot, black-spot scab and rust. The last is probably derived from the German name, "Rostflecken," and should never be used, since there are true rusts of the apple. "Black spot" is the name most used in England, Australia and South Africa, while "scab" is the common name most frequently employed in America. It is apparently the production of scab-like spots on the fruit, which suggested the common name. The disease should not be confused with the scab diseases of other hosts, which are produced by entirely different organisms. Mention may be made of the common scab of potatoes due to bacteria, powdery scab of potatoes caused by a chytrid and citrus scab and peach scab, two entirely distinct fungous diseases.

History.—Scab has been known to botanists since the first part of the nineteenth century, and the causal organism was first described and named by Fries in Sweden in 1819. It was first reported in Germany by Wallroth in 1833. Schweinitz gives the first authentic record of the disease in America, recording its presence in New York and Pennsylvania in 1834, while Curtis mentions it as common in North Carolina as early as 1867. The earliest report from England seems to have been that of Berkeley in 1855. Experiments on the control of the disease in America were started under the direction of Galloway of the U. S. Department of Agriculture in 1886 and 1887, and by 1891 Bordeaux mixture was reported to be the most efficient fungicide. Since that time the disease has been treated by numerous writers and a voluminous literature has accumulated. In 1914 Morris listed 505 different publications dealing with some phase of scab. Much of the published material added but little to our knowledge of the disease, the bulk of the work being empirical tests of various fungicides, or merely spraying demonstrations. Several of the more important researches which contributed to the advancement of our knowledge were the work of Aderhold in Germany (1894 and later) and of Clinton in Illinois (1898). Both workers demonstrated the presence of an overwintering stage of the scab fungus on the fallen leaves, and made

other additional contributions to the life history of the causal organism. Bordeaux continued to be the standard fungicide for scab for many years, but in 1908 Cordley first reported the successful use of lime sulphur, and this soon came into general use in Oregon and Washington, while its value in the East was established by the work of Wallace in New York (1909–1911) and by Scott for the U. S. Department of Agriculture (1909–1911). Since that time lime sulphur has come into general use for the scab disease in the United States, except in regions where it causes serious injury, and the use of Bordeaux has been very largely discontinued. During recent years two phases have been very prominent in the published literature: (*a*) epidemiology (Keitt and Jones, 1926; Wilson, 1928, *et al.*) and (*b*) control. This latter phase has continued to offer a fertile field for reports from nearly all plant pathologists in scab-infested regions.

Geographic Distribution.—Apple scab is prevalent in severe form in all the European countries where the apple is grown and has been sufficiently severe in Australia, New Zealand and South Africa to demand control measures. It is probably of foreign origin and occurred on the wild parents of the apple, accompanying that host ever since it has been under cultivation. It undoubtedly came to America from some European importations. At the present time scab is present in all the states from the Atlantic to the Pacific as well as in British Columbia and the eastern provinces of Canada. The importance of the disease may be evidenced by the fact that nearly all of the Agricultural Experiment Stations in the northern half of the United States have issued one or more bulletins dealing with the character of the disease and its method of prevention. It is undoubtedly the most serious apple disease in the northern portion of the United States and southern Canada, while other diseases like bitter rot and blotch replace it somewhat in importance in the southern range. In the earlier days of Nebraska orcharding, the scab disease of apples was absent and horticulturists at first believed that the mecca for apple growers had been found, but the disease soon appeared and developed with its customary virulence. None of the important apple sections east of the Rockies are now free from the disease, the region of severest infections including eastern Nebraska, Iowa, northern Illinois, Indiana, Ohio, southern Minnesota, Wisconsin, Michigan and Ontario, Pennsylvania, New York and the apple sections of the New England states as well as Nova Scotia. The disease manifests unusual severity in the humid coast regions of the Pacific Northwest, including Oregon, Washington and British Columbia but is somewhat more variable in its occurrence in the Inland Empire country of Washington and Idaho and has almost disappeared from this section during recent years. It is severe in the famous Bitter Root Valley of Montana at an elevation of 3000 to 4000 feet. Scab also finds favorable conditions for its development in the mountain sections of the southern states, especially Virginia and Arkansas. There are but few important apple-growing regions in the United States in which scab is either absent entirely or present in such slight amounts as to render control measures unnecessary. The most important of these favored regions are the famous Yakima and Wenatchee valleys of central Washington. In both of these regions the growers have not yet found it necessary to spray for scab. Cultural practices and climatic conditions in these irrigated valleys have been unfavorable for the spread of the scab disease, and it seems probable that the trouble there will never become an important factor in apple production.

Symptoms.—Scab upon the *leaves* may show definite, more or less circular spots which are brownish or gray in the early part of the season, but later, at least under favorable conditions, may become even olive green or nearly black. These spots when young frequently show the radiating dendritic ramifications of the scab fungus, easily visible with a

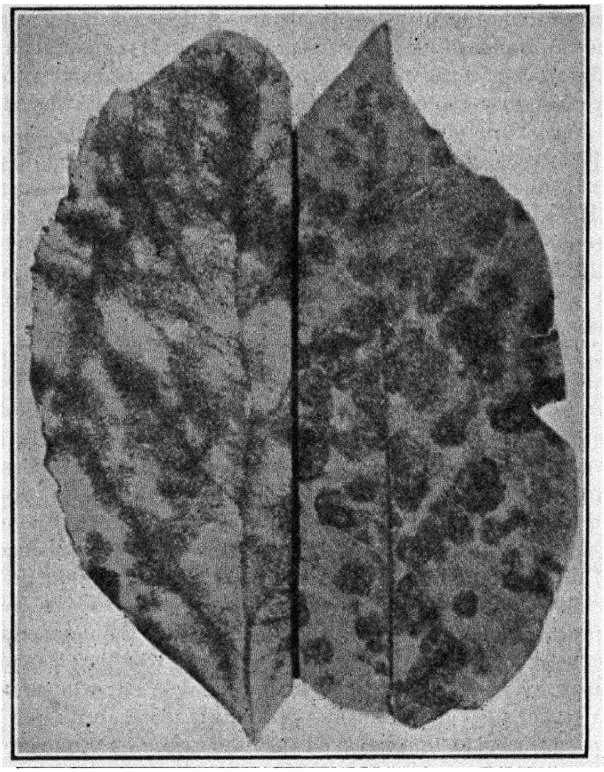

FIG. 176.—Apple leaves showing heavy scab infections, diffuse or spreading type on the left, localized spots on the right. (*After Wallace, Cornell Univ. Bul.* **335.**)

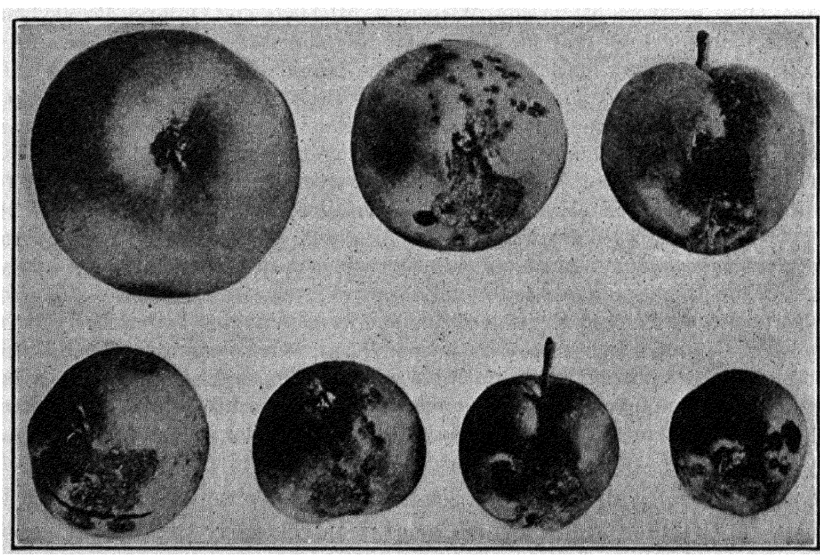

FIG. 177.—A normal apple and others variously spotted, deformed or atrophied as a result of a severe attack of scab.

hand lens. Later in the season the scab spots on leaves not affected sufficiently to fall from the tree may show considerable thickening of the leaf tissue, the brown, velvety surface becomes smoother and dull and occasionally dead areas drop out leaving irregular perforations. Under certain conditions the fungus spreads diffusely over the surfaces of the leaves, causing a rather uniform brown coloration. When the scab spots are abundant or when the diffuse form is severe there may be more or less reduction in the size of the leaves, with curling and distortion. In the severe diffuse type much leaf tissue is killed and the affected leaves or portions of them may appear as if scorched or burned. In some cases an entirely different symptomology may be noted, the affected trees often

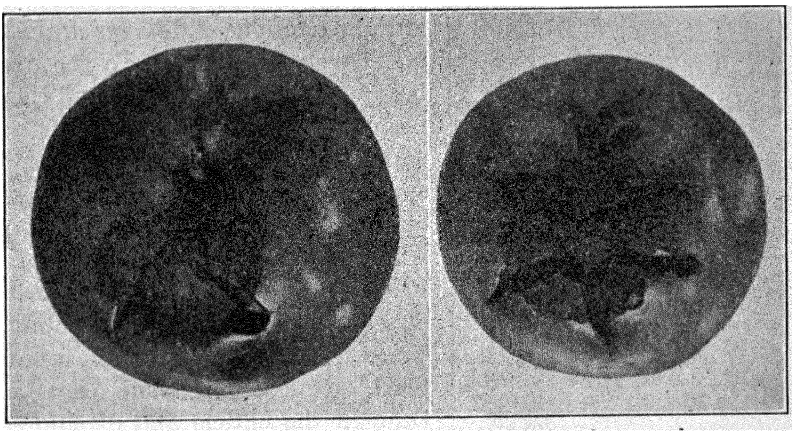

FIG. 178.—Apples with large scab lesions showing deep cracks.

showing much yellow foliage. The leaves may be gradually shed and a partial or almost complete defoliation may result. Some of the affected leaves will be of a uniform yellow color, while others may be more or less variegated with yellow, brown and green. The scab fungus may be found on the isolated green spots, the affected spots showing a conservation of the chlorophyll. The diseased areas may appear on either surface of the leaves, but the earliest infections are most frequently upon the lower surfaces.

The scab lesions on the fruits first show as small, more or less raised, brown or black, somewhat circular spots with no break in the skin. As they increase in size the cuticle ruptures and the exposed surface shows a brown or almost black velvet-like appearance surrounded by an irregular whitish band, which represents the frayed and torn cuticle. In this condition, which is generally reached by early July, the surface of the fruit lesions has much the same appearance as the leaf spots. As the fruit develops, the spots increase slowly in size and the central and older part may become bare and brown and show a more corky character, while the dark peripheral zone and the band of cuticle still persist. Still later

in the season the cuticular band may disappear entirely, and the whole scab lesion becomes slightly raised, and shows a rough corky surface, not at all resembling the early or midseason appearance. Scab spots may be few in number or they may be sufficiently numerous to coalesce and form extended lesions. Severe infections are likely to cause more or less cracking of the fruit.

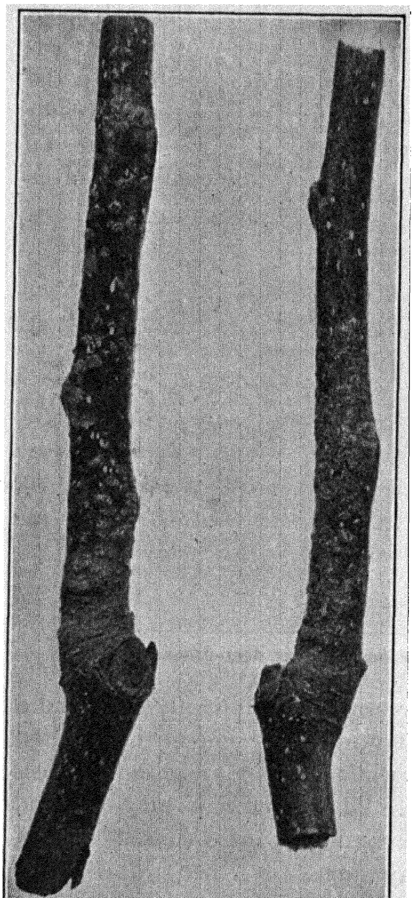

FIG. 179.—One-year-old apple sprouts showing numerous scab lesions.

The above is the normal or typical development of the scab spots. In late-autumn infections they are generally darker, being dense and black, and do not break through the cuticle as soon as the early infections. These autumnal lesions seldom reach the stage of development when the center becomes bare and brown, and they are frequently much smaller than the spring infections. For this reason the spots of this type are sometimes designated as "pin-point scab." Apples which are apparently free from scab when they go into storage sometimes develop infections of the pin-point type. These infections may remain as covered black spots or in some cases break through the cuticle and expand somewhat. *Storage scab* may result from incipient infections that were too small to be seen at picking time, or there may be new infections during storage from conidia originating from some of the older scab spots.

Scab lesions may occur on the blossoms during favorable years. These appear generally on the pedicels, calyx or the young fruits about the time the petals fall. There may be single small lesions or the diffuse type of infections may predominate.

Scab spots may appear on the twigs, being confined to the bark of 1-year-old shoots. Twig infections were formerly considered rare for the apple in America, but they have been observed in Maine, New York, New Jersey, Nova Scotia, Washington and Montana. Young lesions on the twigs are very similar in appearance to those on the fruits, showing the central spore surface bordered by the uplifted epidermis. Later in the season the spores may disappear and the bark show a more scaly character,

due to the peeling of the bark in flakes. This type of injury is more common on pear twigs than on apple and has been called "grind or scurf" by German writers. The twig lesions may be few and scattered and thus may be easily overlooked, or they may be so numerous as to coalesce and produce more extended affected areas.

Effects.—Scab offers an illustration of a disease which may cause a serious loss without endangering the life of the tree. This is due to a lowering of both quality and quantity of the commercial product, the fruit. The injury may be current, that is, affecting the immediate production, or it may be in part delayed till the season following a serious epiphytotic.

As a result of the foliage attacks there may be more or less defoliation. Many leaves showing severe scab of the diffuse type or numerous distinct lesions fall from the trees in early summer. Many of the affected leaves, however, persist through the growing season, but they are only partially effective in carrying out their normal work of food manufacture, since their producing power is lessened in proportion to the amount of tissue (surface area) killed by the scab fungus. Severe diffuse forms of scab may kill extended areas of leaf tissue and in epidemic years many leaves appear as if scorched or burned. Severe foliage infections must, therefore, have a pronounced devitalizing effect by lessening the available carbohydrate food, and influence to some extent "the formation of fruit buds for the following year and hinder the normal wood growth which is the basis for future crops."

It is by direct attacks on blossoms or fruit that scab takes its largest toll. Severe infections on blossoms, fruit pedicels or young fruits cause a complete blighting or dropping of the fruits while still very small. Since this phase of scab is more obscure than the others, and thus escapes their notice, orchardists frequently attribute this failure to set fruit to adverse climatic conditions, such as frosts or cold rains during blossoming time. Authentic cases are on record in which the apple crop was a complete failure due to the destruction of blossoms and young fruit by scab, but such a wholesale destruction is not very common even in unsprayed orchards. In ordinary scab years, however, the standard spraying practices increase the set of fruit, showing that the disease is nearly always causing some blighting of blossoms and young fruit.

Fruits that escape the early severe attacks may persist on the trees through the growing season, though exhibiting one to numerous lesions. According to the degree of infection, the affected fruits may be reduced in size or variously deformed, but even though normal size may be attained the presence of the scab lesions is a disfiguring feature which cannot be overlooked. In the fall there may be a premature dropping of scabby fruit, but it is difficult to say just how much of the dropping is due to scab. Fruits which show large lesions, or numerous ones which coalesce,

frequently develop rather deep cracks or fissures extending into the pulp. These are especially pronounced in such susceptible varieties as the Early Harvest and McIntosh. All infected fruits must be sorted out and placed in a lower grade and sold for less than the clean fruit.

The effect of scab on the keeping qualities of the fruit must not be overlooked. Scabby fruit shrivels in storage faster than normal fruit, since the breaks in the skin caused by the fungous lesions increase the water loss. The losses from rotting are increased by scab, since the lesions offer easy avenues for the entrance of various rot-producing fungi. Cases are on record in which the development of scab in storage has caused heavy loss, since it made necessary a resorting and packing. The careful culling out of infected fruits at packing time is an added burden and expense to the grower.

Losses from Scab.—The aggregate loss from scab each year is enormous and even on the most conservative estimates runs into millions of dollars. The loss to be charged to scab is due to: first, the reduction in the total yield of the country; second, the lowering of quality and consequently a reduction in the financial returns to the orchardists; and, third, to the increased cost of production due to the necessity for carrying out control measures and the increased labor of sorting and packing. Spraying is not a perfect control of the disease and besides there are many small home orchards in which spraying is not practiced. It has been estimated that there is an average annual loss of more than $40,000,000 as a result of failure to spray apples. Of course, this should not all be charged to scab, since other troubles like blotch, bitter rot, rust, etc., are also controllable by spraying.

A number of specific instances will serve to emphasize the loss from scab and show the necessity for practicing the known control measures. It is estimated that the average annual loss from scab in New York State is not less than $3,000,000; that the damage done in Illinois amounted to $6,000,000 in a single year; that there was a loss of 15 per cent of the crop in Montana in 1911 and 1912; while $48.50 per acre is given as the average annual loss in Australia. A loss of $47 per acre has been estimated as a result of failure to spray for scab in New York State, while Minnesota records place the loss for that state at $51.40 per acre. Figures of this sort should serve as an incentive to induce growers to take more pains in the work of control.

Etiology.—Scab is due to an ascomycetous fungus, *Venturia inæqualis* (Cke.) Wint., which lives as a parasite on the leaves, twigs and fruits of the apple (the imperfect or Fusicladium stage), and completes its life cycle as a saprophyte (the perfect, perithecial or Venturia stage) upon the dead and fallen leaves of the same host.

All of the publications on scab in America dealt entirely with the parasitic or Fusicladium stage (*Fusicladium dendriticum*) until the work

of Clinton in 1901, when attention was directed to the saprophytic stage on the overwintering leaves, and the connection between the two definitely established. The connection had long been suspected (as early as 1887), but definite proof of the relationship was first published in the exhaustive researches of Aderhold in 1894 and later. The earlier work of the German botanists was at first overlooked and the conclusions of Clinton were arrived at without a knowledge of their discoveries. The work of Clinton and Aderhold has been confirmed and extended by other observers in this country and in Europe. Some of the most important

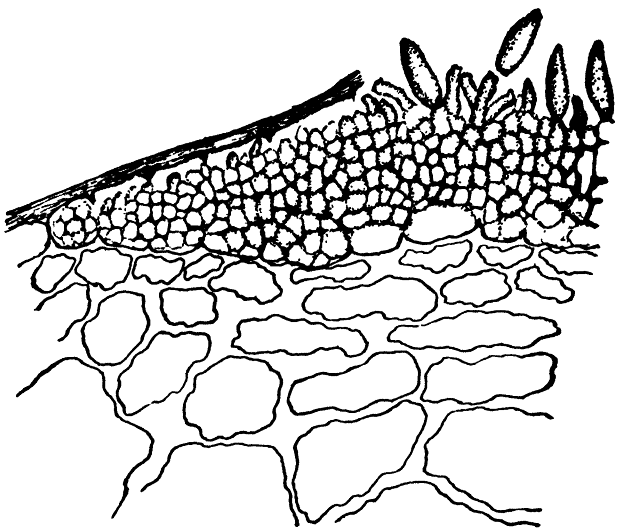

FIG. 180.—Conidial stage of *Venturia inæqualis*. Section from the edge of a fruit lesion, showing uplifted cuticle, fungous stroma, conidiophores and conidia. (*After Wallace, Cornell Univ. Bul.* **335**.)

facts which have supported the relationship of the two forms are as follows:

1. The constant association of the perithecia with leaf lesions known to represent the parasitic stage during the previous season.

2. The development of the Venturia mycelium within the fallen leaves directly from the stromata of the Fusicladium stage.

3. The typical Fusicladium stage resulting from artificial inoculations made with ascospores.

4. Production of typical Fusicladium mycelium and characteristic summer spores of the scab from cultures started from ascospores on artificial media.

5. Production of perithecia of Venturia in artificial cultures originating from the Fusicladium stage.

6. The relation of the first spring infections of scab to the period of expulsion of the ascospores.

The mycelium on the leaves is at first nearly hyaline, subcuticular and develops radially with a characteristic dendritic branching which suggests the specific name applied to the conidial stage. This mycelium later develops one or more layers of closely aggregated cells, forming a stroma-like growth which breaks the cuticle and permits the production of numerous erect conidiophores exposed to the surface. The older mycelium and conidiophores are brownish and the numerous spores and

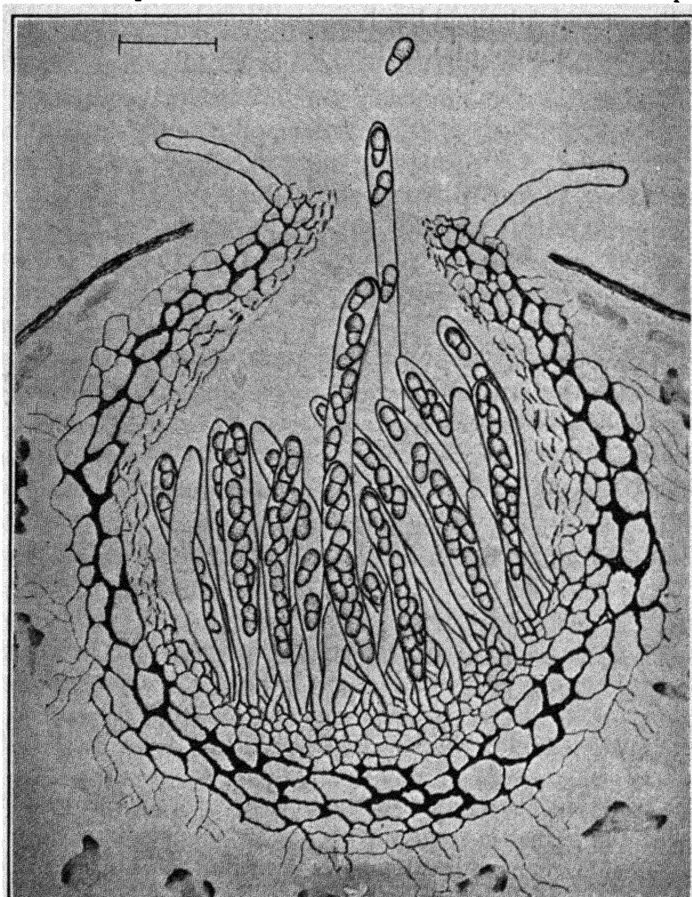

FIG. 181.—Section of a perithecium from an overwintered fallen leaf. (*After Wallace, Cornell Univ. Bul.* **335**.)

conidiophores give the scab spots a powdery or velvety appearance and an olivaceous or almost black color. On the fruit the stromatic cushions of mycelium frequently completely destroy the epidermal cells and extend deeper into the pulp tissue. The conidiophores are short, erect, straight when young, but more or less flexuous with age, olivaceous and continuous or one- or two-septate. The conidia are lanceolate or ovate, with the base generally somewhat truncate, continuous or more rarely

one-septate, 12 to 22 by 6 to 9μ, and are produced in indefinite numbers from each conidiophore. The conidia are "resistant to detachment from the conidiophores when dry but quickly become detached in water." For this reason their chief mode of dissemination is meteoric water.

While the conidial or Fusicladium stage is produced on all parts of the host, the ascigerous or perithecial stage is formed only on the fallen overwintering leaves. The mycelium develops as a saprophyte on the dead fallen leaves, and penetrates into the mesophyll from the superficial stromatic layers. The perithecia appear mostly on the under surface as minute black dots or pimples barely visible to the naked eye, but are really spherical or subspherical bodies 90 to 100μ in diameter embedded in the leaf tissue and protruding on the surface as minute dome-shaped pimples. The mature perithecium is ostiolate and the short beak is generally armed with several short bristles; the wall is dark and composed of several layers of cells; and each produces an indefinite number of oblong spatulate asci or spore sacs 55 to 75 by 6 to 12μ. The asci are eight-spored, the ascospores two-celled, 11 to 15 by 5 to 7μ, olive brown, with the upper cell wider than the lower cell. Paraphyses are lacking.

The perithecia on the fallen overwintered leaves begin their development in the fall and early winter, and mature ascospores are ready to be expelled by the time the petals show pink (often earlier). The spores are forcibly expelled following the warm spring rains. Each ascus elongates, protrudes its tip through the ostiole, bursts with the forcible expulsion of its spore content and then the process is repeated by other asci until all the asci are discharged. The period of ascospore expulsion varies in different localities. On the Pacific Coast the first expulsion occurs in late February or early in March and discharge continues for about 3 months before all the perithecia are exhausted. This means that ascospores are expelled before the foliage has even started. In the eastern United States (New York) ascospore expulsion does not begin so early and according to available data does not continue for much longer than 1 month. Recent studies of ascospore discharge in Delaware (Adams, 1925) and in Virginia (Schneiderhan and Fromme, 1924) have shown conditions more comparable to those of the Pacific Coast. Spore discharge begins very soon after the beginning of rainfall. In Virginia, in 1922, 16 discharge periods were recorded, beginning on Apr. 18 and ending June 12, while in 1923 there was a total of 13 discharges from Apr. 28 to July 30. The studies in Delaware emphasize the great variation in the time of initiation of spore discharges and the length of the period during which discharge continues. For example, in one locality discharge began about May 19 and continued for little more than a month, while in a test made in another county during the same season the discharge began on Mar. 28 and continued into the month of June. Studies in Wisconsin have shown, in general, a shorter duration of spore

discharge more comparable to the New York experiences: At Madison beginning Apr. 23 and ending June 12, while at Sturgeon Bay the first ascospores were discharged on May 20 and the last on June 30 (Keitt, 1921). The time of ascospore discharge has a bearing on the success of spraying. Recent studies have emphasized that it is difficult to time scab sprays correctly. According to Adams (1925), although conditions favoring spore discharge are beyond one's control, they can be predicted to a certain extent. "However, it is not possible or practical to attempt spraying on the prediction of weather conditions that may favor all periods of possible spore discharge." In other localities (Young and May, 1927) very satisfactory results have been obtained by timing the sprays in accordance with the reports of the local weather service. Ascospore expulsion is controlled by moisture and temperature conditions, and consequently we may expect variations in different regions. While it is a common thing to find abundant discharge of spores about the time the blossoms show pink, activity may begin earlier or it may even be delayed until after the blossoms have opened. In regions or seasons of early spore expulsion, scab is likely to be severe, while a delayed expulsion may lessen the amount of the disease that develops.

The expelled ascospores are carried upward by wind or air currents and reach the young leaves or blossom buds and produce the first infections after a period of incubation. These are the primary infections and they soon give rise to conidia which may be scattered to other host parts and give rise to secondary infections.

The primary lesions from the ascospores furnish conidia which may cause very abundant secondary infections on fruit and leaves. Primary or secondary lesions may furnish conidia throughout the growing season and new infections may result whenever moisture and temperature conditions are favorable, but it should be understood that young leaves and fruits are much more readily infected than older and more mature structures.

Late infections may develop during some seasons and they are likely in those regions of abundant rain or fog and moderate temperatures. Apples that were free from scab at the time of picking but were taken from scabby orchards may develop infections after they go into storage. It seems probable that infection occurred in such cases in the orchard, and that the incubation period had not expired at the time of picking.

The life history of the scab fungus may be summarized as follows:

Scab infected leaves fall to the ground in the autumn, and the overwintering structures, the *perithecia*, develop within the leaves from a saprophytic mycelium which originates from the superficial stromatic cushions. The perithecia mature their ascospores in the early spring, and these are forcibly expelled following the warm spring rains and produce the first or primary lesions which soon produce conidia; these conidia

are scattered by wind or rain and produce secondary infections, and these secondary lesions are, in turn, the source of other conidia, from which still other infections may arise.

The part which twig infections play in carrying the fungus through the winter period is rather uncertain. There is some evidence that in the milder climates twig lesions may resume spore production in the spring, but all evidence points to the ascospores as the important source of the primary spring infections in most scab-infested regions.

Predisposing Factors.—The abundance and severity of scab in any locality are dependent on the proper combination of climatic factors, especially temperature and moisture, which influence the development and expulsion of ascospores, the primary infections by ascospores and secondary infections by conidia. The temperatures of January, February and March are believed by some to be more important to ascospore development than the amount of precipitation in the period immediately preceding the maturity of the ascospores. Germination of ascospores or conidia may take place from 0.5 to 32°C., with infection resulting at temperatures ranging from 6 to 26°C. Continuous wetting of surfaces for a certain length of time is necessary for infection, the period of wetting required being longer for the low than for the higher temperatures. During the periods of low temperatures, it seems that the lengths of time that susceptible surfaces are held moist will be more likely to be those required for infection than when the temperatures are relatively high. In general, it can be said that cool, moist weather is favorable, while higher temperatures are unfavorable even though moist conditions prevail. Any time after primary or secondary sources of infection are available, a cool rain period followed by conditions which favor the retention of moisture is very favorable to infection. Showers followed by winds are unfavorable, since the moisture evaporates rapidly, while showers in the evening followed by a calm night are very conducive to infection. The relatively high spring temperatures and bright sunshine of some of our important apple districts, like the Yakima and Wenatchee valleys of Washington, are probably the principal factors which have excluded scab.

It should also be noted that poorly pruned trees with dense branching and heavy foliage generally suffer more than well-cared-for orchards with open growth which permits a good circulation of air and rapid evaporation of moisture. Trees in low pockets are more subject to scab than those on hill tops where the air drainage and evaporation are good.

Host Relations and Varietal Resistance.—The conidial stage of *Venturia inæqualis* occurs on many *Pyrus* species of the *Malus* group, but the scab of hawthorn (Cratægus) and Sorbus represent distinct species which cannot infect apple varieties. The scab of pears (*Venturia pyrina*) agrees, in general, in its life history with the scab of apples, but here also

cross-infections are impossible and the fungus represents a distinct species. The much more frequent occurrence of twig lesions in pear scab is one of the striking differences.

No varieties of apples are immune to scab, but great variation in susceptibility is shown. It is found that certain varieties may be resistant 1 year and heavily scabbed another year, under conditions favorable for scab. It is also to be noted that varieties noted as resistant in one locality may prove very susceptible in another region. Various writers have listed varieties as resistant, moderately resistant, moderately susceptible or susceptible, but it may be noted that the Baldwin, which is generally classed as resistant, has shown as high as 98 per cent scab. No attempt will be made to explain this and many other similar variations. The general reputation of a variety can, however, be taken into consideration in making selections for commercial orchards. In the light of recent studies on other parasites and work of Wiesmann (1931) it seems probable that physiological strains may explain the variable reports on resistance. Five strains of apple scab and four of pear scab are reported differing in cultural and morphological characters and possibly in pathogenicity.

Control.—While the use of fungicides constitutes the principal means of protecting orchards from scab, there are certain other practices that should receive attention.

Planting Practices.—Since scab sprays are applied with reference to time of blossoming, it is important that commercial plantings should consist of solid blocks of certain varieties rather than of mixed plantings. Even in the small home orchard the mixed planting of varieties makes timely spraying doubly difficult. A recognition of the predisposing influence of pockets or lowlands with poor air drainage would suggest the selection of orchard sites to avoid this factor.

Sanitary Measures.—Observations in many sections agree in reporting scab at its worst in neglected orchards. Failure to keep the trees properly pruned makes a dense growth of branches and heavy foliage, which excludes sunshine and retards evaporation, so that spore germination, and consequently infection, are favored. If there were no other reasons for pruning, the effect of failure to do so upon scab alone would be sufficient argument for attention to this orchard practice.

Our knowledge of the part which hibernation of the scab fungus on fallen leaves plays in the life history of the disease suggests that their destruction would lessen the primary infections. Clean cultivation and close mowing of cover crops or weeds, either to bury the leaves or to give less favorable conditions for the development of perithecia and ascospore dissemination, are to be looked upon as measures to supplement the use of fungicides.

Use of Fungicides.—The carrier for the toxic chemical may be either water (spraying) or air (dusting). Spraying generally gives better control

and is more generally practiced. The time of application of the fungicide may be based on the stage of growth of the trees or upon the maturing of the ascospores in connection with reports by a local weather service if such spray warnings are issued. The following table will give some of the various applications recommended, beginning with schedule A, which is designed to meet the needs when scab is epidemic and susceptible varieties are concerned. The other schedules are designed to meet the needs under less severe conditions, either seasonal or environmental.

SCHEDULE FOR SPRAYING OR DUSTING FOR SCAB

A	B	C	D	E	F	G	H	I	J	Relative value, per cent
1. Green tip stage	1									
2. Early closed cluster	..	2	2	..	2	..	2	..	..	40
3. Open cluster spray	3	3	3	3	3	3	..	3		
4. Calyx (2/3 to 3/4 petals off)	4	4	4	4	4	4	4	4	4	40
5. Ten days to 2 weeks later	5	5	5	5	..	5	..	..	..	10
6. Summer sprays at selected time in 4 weeks or more after calyx	6	6	..	6	..	..	..	..	..	10

Conditions are so variable in different regions that the advice of local authorities should be followed as to the spray program to be adopted and the fungicide to be used (also the insecticide).

The following are the more important sprays which have been used:

1. Bordeaux, at strengths varying from 4–4–50 to 1½–2–50[1] or 2–10–50.
2. Liquid lime sulphur, 1 gallon to 40 to 50 gallons of water.
3. Dry lime sulphur, 3–50 or 4–50.
4. Dry-mix lime sulphur (see Brown Rot, p. 522).
5. Calcium sulphide, 8–50 to 12–50 (Hurt and Schneiderhan, 1929).

The dust mixtures which have given most general satisfaction are:

1. Finely powdered or dusting sulphur 85 to 90 per cent plus 15 or 10 per cent fluffy, powdered lead arsenate, using 1¼ to 2½ pounds per tree according to size.

2. Dusting sulphur 40 per cent, lead arsenate 20 per cent and 40 per cent hydrated lime, the latter used as a filler.

3. Hydrated lime 86 per cent, dehydrated copper sulphate 10 per cent and calcium arsenate 4 per cent (Sanders' dust).

The period up to 1908–1911 when lime sulphur first came into use has been designated the Bordeaux and Paris-green period in apple

[1] Even as weak as 0.5–2.5–50 has been reported to give as good control as 3–9–50 (Ballou and Lewis, 1927).

spraying, since this combination of fungicide and insecticide was generally used. Repeated experiments continuing up to the present have shown the superior fungicidal value of Bordeaux over lime sulphur for scab control, but because of the less injury to foliage and fruit, lime sulphur has come to be generally used especially in America, except in regions where lime-sulphur injury is extreme (see Bordeaux Injury, p. 223; Lime-sulphur Injury, p. 230) or where certain other diseases must be controlled for which lime sulphur is not so effective.

In earlier tests dry lime sulphur gave poorer control than liquid lime sulphur, but in more recent trials it has given protection equal to the liquid lime sulphur (Britton *et al.*, 1921; Doran and Osmun, 1924; Keitt and Jones, 1926; Ballou and Lewis, 1927). Dry-mix lime sulphur and calcium sulphide have yielded results equal to any other sulphur-containing sprays and with less injury (Hurt and Schneiderhan, 1929). Floatation sulphur has given adequate control (see Brown Rot, p. 522). According to Dutton (1930), dry-mix lime sulphur, wettable sulphur, colloidal sulphur, activated sulphur and sulfocide have proved inferior to liquid lime sulphur. Proprietary remedies such as Adheso, B.T.S., Spra-sulfur, Fungi-Bordo and Soluble Sulphur are not to be recommended, either because of injury to fruit and foliage or because of greater cost.

Earlier tests of the dusting method gave very poor results, but since the employment of finely divided dusts and improved dusters, this method of control has given results in some sections that have justified its use. It is now generally conceded that dusting is somewhat inferior to spraying (Folsom, 1926; Giddings *et al.*, 1927; Moore, 1930; Hamilton, 1931

Spraying of fallen leaves to prevent the formation of ascospores has been tried with promising results. Curtis (1924) obtained as much as 40 per cent reduction from three dead-leaf sprays. The use of calcium arsenite for this purpose has also been suggested.

Does It Pay to Spray for Scab?—Spraying must be looked upon as an insurance, and growers must decide from results whether it is a paying proposition. There are at least three types of localities, so far as scab control is concerned:

1. Those in which scab is unknown or relatively rare. In such localities spraying would never be profitable.

2. Regions in which severe scab years are infrequent. There are places where scab may be severe one season out of four or five. In such cases the actual money value of yearly spraying is doubtful.

3. Regions in which scab-free years are uncertain and relatively infrequent. In such localities the successful orchardist must spray each year. Careful cost and production records have shown net profits, due to spraying, of $65 to $400 per acre.

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BLACK ROT, CANKER AND LEAF SPOT

Physalospora malorum (Berk.) Shear

This is a fungous disease of apples, pears, quinces and numerous other hosts, being best known for its ravages in apple orchards of the eastern United States. In its various phases it attacks the fruit as it is approach-

ing maturity or in storage, causing a fruit rot; the twigs and limbs, causing *blight* and *cankers;* and the leaves, causing a characteristic *leaf spot.* The fruit rot was first studied and various names descriptive of the symptoms were applied, as black rot, ring rot, blossom-end rot and brown rot. The phase of the disease on the twigs and limbs has been described as dieback, twig blight, apple canker, black-rot canker and the New York apple-tree canker. The leaf attacks are referred to as leaf spot, leaf blight, brown spot and frog eye.

History.—Although the causal fungus had previously been known, the first report of the occurrence of black rot as a disease of apples was by Peck in New York in 1879. This was still the only phase of the disease that had been definitely recognized as due to *Sphæropsis malorum,* until 1898 when Paddock published his first report on New York apple-tree canker and showed that a canker of apple common in New York and adjacent territory was due to the same organism as the fruit rot. Previous to the recognition of the canker phase of the disease, leaf spots of the apple were attracting attention. The first consideration of the economic aspect of leaf spot was by Alwood in 1892, but he attributed it to an entirely different fungus, *Phyllosticta pyrina,* which was found fruiting on the spots. Another Phyllosticta species (*P. limitata*) was reported by Stewart (1896) as the cause of a serious outbreak of leaf spot on Long Island. The idea seemed to prevail for some time that Phyllosticta species were the most important agents in the production of apple leaf spots, although other fungi were reported as accompaniments. The question of the parasitism of the two Phyllosticta species was taken up by Stewart and Eustace (1902) and they decided that "at least a large part of the so-called apple leaf spot is due to spray injury and weather conditions and not to fungous origin." The fungi were believed by them to be saprophytes which invaded the tissues killed by other agencies. Alwood had found *S. malorum* in leaf spots as early as 1898, while Clinton (1902) recorded this pathogene as the cause of a leaf spotting of the apple in Illinois, and later (1903) recorded this pathogene as the cause of most of the leaf spot of the apple in Connecticut. Studies by a number of workers about the same time (Scott and Quaintance, 1907; Lewis, 1908; Hartley, 1908) began to throw doubt on the parasitism of the fungi associated with leaf spot, while the large number of species isolated and the unsuccessful inoculations in the studies pointed to the saprophytic relations of associated organisms. While positive evidence was lacking, Lewis (1908) decided that "the fungus, *Sphæropsis malorum,* which is known to cause canker of apple limbs and is an active parasite, will be found to be the primary cause of apple leaf spot." It remained for Scott and Rorer (1908) to prove definitely by inoculations that the common leaf spot as it occurred in the Middle West was due to the black-rot organism and that most of the accompanying fungi, including *P. pyrina,* probably occurred on leaf spots only as saprophytes.

Later studies by various workers confirmed the first discovery of the apple rot and showed that the pathogene would affect maturing apples in the orchard, and was also less important as a disease of pears and quinces. The continued study of the canker phase of the disease emphasized its importance, definitely established the connection between the organism in the fruit and in the cankers and yielded further important contributions on the etiology of the disease. The ascus stage has been studied by Hesler (1912), Shear (1914), Cunningham (1923) and Shear *et al.* (1924), and all have agreed that it is a Physalospora, but doubt exists as to its identity with *P. cydoniæ* (Arnaud, 1912). In the studies of the leaf-spot phase of the disease it was soon noted that the spots so prevalent in the spring later enlarge to produce the condition known as "frog eye," especially in the southern states. This condition was attributed by

some to other fungi or to other fungi following the Sphæropsis infections. Roberts (1914) was able to produce typical frog-eye spots by inoculating dead spots with an Alternaria. From his results and the work of others it seems that the frog-eye development may be due to Sphæropsis working alone or in association with some other organisms.

Early in the history of black rot, attention was given to control measures, but as late as 1890 Scribner wrote: "Nor can we at this time propose any treatment which would be likely to be effective and at the same time practical." Spraying was suggested, but the first carefully controlled experiments for the protection of the fruit were reported by Wolf (1913). Spraying was recommended for leaf spot before the relation to Sphæropis was established, the results coming largely from spraying for the control of scab and bitter rot. Various workers since 1901 have reported a successful control of leaf spot. Mention should be made of special experimental work on control by Crabill (1915) in Virginia and Walton (1920) in Pennsylvania.

The most complete study of black rot in its various phases was published by Hesler in 1916. Later work has been concerned largely with control or establishing the presence of the disease in new territory.

Geographic Distribution.—The disease is widely distributed in America from the Gulf states northward to Ontario, Quebec and Nova Scotia. While it has been reported from California, Oregon and Washington, it seems to be a rare trouble west of the Rocky Mountains. The various phases of the disease have not been equally prevalent throughout its range. The canker has been especially prominent in New York and adjacent territory. In the extreme East, the Ozarks and Virginia, the fruit rot and the leaf spot have been the general phases of the trouble. "Speaking generally for northeastern America, the Middle West and southeastern Canada, this apple disease in one or more of its three forms stands second only to apple scab in importance" (Hesler and Whetzel, 1917). Black rot occurs in Europe from Italy and France northward to Germany and England, and extends eastward into southern Russia. It occurs also in Australia and South Africa and has more recently been reported from New Zealand. The disease is of minor importance in European countries, and in New Zealand the principal damage is from the canker phase of the disease.

Symptoms and Effects.—The *leaf-spot* phase of the disease begins soon after the leaves unfold from the bud. The young lesions show first as minute purple specks, which soon enlarge to circular spots, 2 to 10 millimeters in diameter, with an average of 4 or 5 millimeters. The tissue of the lesion gradually becomes brown, and the spot sharp in outline, with a faintly raised margin. As the tissue dries out, the spot may become a dirty-gray color and in numerous cases minute black fruiting bodies may be seen occupying the upper surface (mostly saprophytic intruders). Bordeaux injury or localized killing from free arsenic may be responsible for leaf lesions which closely resemble these black-rot leaf spots. While this is the type of the leaf spot that predominates in certain localities, in others they deviate to such an extent that at first they were supposed to be due to another organism. Some of the spots remain in the condition already described for the entire season, but many after ceasing growth for a time become active again.

From one or more points on its margin the spot begins to spread, forming brown crescents much darker than the older portion of the spot. These new

brown crescents are always bordered with purple on the outer edge. They extend outward and sideways until they finally come together, completing the circle of brown around the gray center. More crescents of brown develop from this first brown circle, growing together and forming a second brown circle. This formation of concentric circles continues until the leaves fall in the autumn or as a result of the injury, the older rings become lighter in color with age. The spot at the time of leaf fall is a more or less irregular blotch made up of a light-gray center, sharply defined, surrounded by many concentric rings of brown with light zones between (Crabill, 1915).

This is the condition which has suggested the name "frog eye." The size of the spots depends on the weather conditions and also upon the number of infections per leaf, the maximum size of about 1 inch in

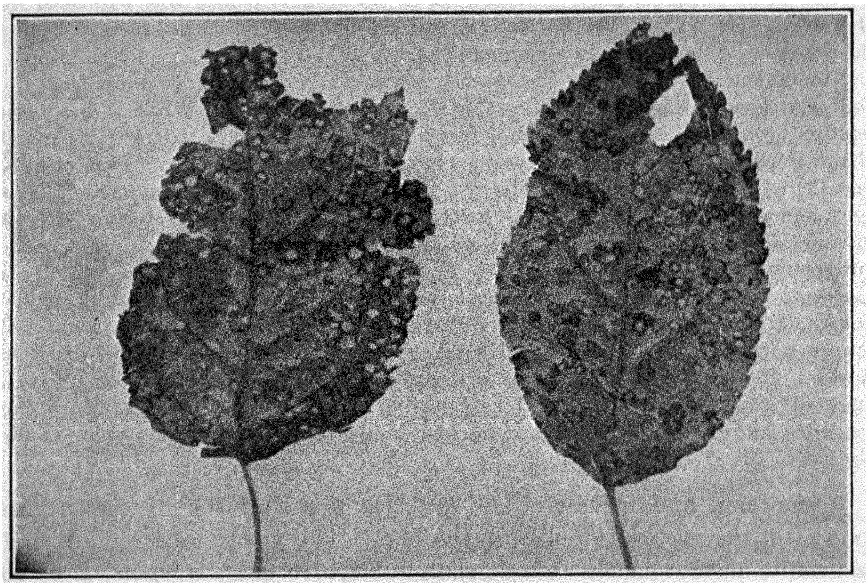

FIG. 182.—Apple leaves showing leaf spots caused by black-rot fungus (*Physalospora malorum*). (*After Scott and Rorer, Bur. Pl. Ind. Bul.* **121**.)

diameter being reached under the most favorable conditions. The spots may be few in number or they may be numerous and adjacent lesions may coalesce to form more extended dead areas.

The *fruit rot* is not in evidence until a few weeks before maturity or later as a storage rot. Fruit lesions are first apparent as small brown spots, frequently at a worm hole, which later darken as they expand in size and finally become black. A single lesion to an apple is the common condition, and it may be anywhere on the surface, but is frequently located at the calyx end. In some cases concentric zones of different shades of brown or black appear, but in others no zonation is apparent. A fruit lesion continues to advance and to penetrate the flesh of the fruit, the invaded tissue having the form of a cone with the external area

of the lesion as its base. Within a few weeks after infection minute black pimples may appear in the skin of the fruit, and these by their numbers contribute to the black coloration. The rot advances until the entire fruit is involved and

. . . later stages in the development of the rot show a shriveled and much wrinkled surface, which typically becomes covered with black pustules. Ultimately a dry mummy is produced, which may hang to the tree for a year or more (Hesler, 1916).

It is this phase of the disease that has suggested the very appropriate name of *black rot*. It has been confused with brown rot (see Chap. XX for difference) and also with bitter rot. It can be easily distinguished

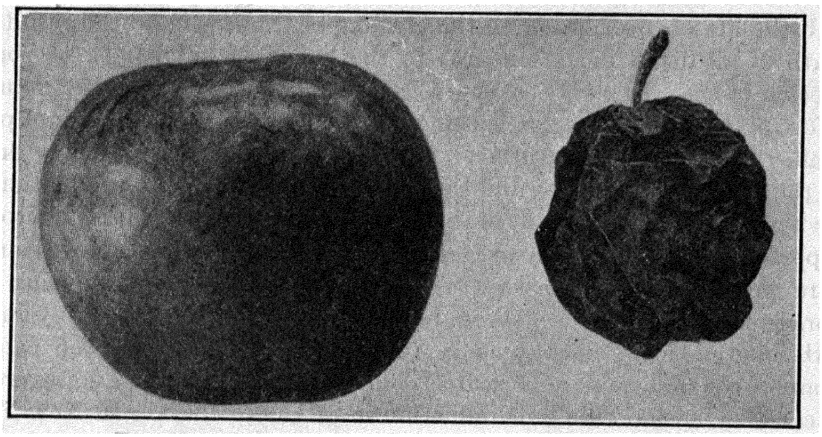

FIG. 183.—Normal apple and shriveled black-rot mummy.

from the latter, which has an unpleasant taste, and pinkish fruiting pustules.

The disease may attack the trunk, larger limbs, smaller branches or even the twigs, producing either localized *cankers* or a *blight* or *dieback*. The typical cankers on the larger limbs, appearing more frequently on their upper surfaces, are first in evidence as reddish-brown discolorations of the bark, which later become slightly sunken as the affected tissue dries. Lesions may remain small and cease to extend at the end of the first season, but in many cases a diseased area continues to enlarge year after year until it has girdled the branch or has extended for a yard or more.

A crevice may appear at the margin, limiting, temporarily at least, the extent of the lesion. Further spread of the pathogene results in the formation of a prominent spot, which soon forms a second line of demarcation between the healthy and diseased tissue. Repetition of this process from one or more points at the margin occurs, thus producing a lobed appearance; or the spreading may arise from all points about the first marginal crack, so that a series of concentric crevices is developed, as described for the frog eye of the leaves. The bark

remains closely appressed to the wood for at least a year; later the dead bark cracks, and falls away, exposing the wood and a callus around the margin of the wound (Hesler, 1916).

The minute, black fruiting pustules may be very abundant over the bark of blighted twigs or in the bark of the localized cankers. Cankers may show only a superficial roughening of the bark, while in other cases the bark is killed to the cambium and the underlying wood may be stained and cracked.

The injury from the black-rot disease is due to the following: (1) the reduced photosynthetic capacities of the spotted foliage and to the early defoliation when spotting is severe; (2) to the rotting of the fruit just previous to maturity and during storage; (3) to the interference of cankers with the life of branches or to the girdling of branches and the resultant death of all distal parts; and (4) to the blighting or dieback of young affected twigs. In severe cases defoliation may occur 6 weeks to 2 months before maturity of the crop, causing the fruit to remain small and of poor quality or to drop prematurely. Such foliage losses make a heavy drain on the vitality of the tree and seriously interfere with the production of the next year. The amount of the fruit rot is variable, but it is a factor of importance from Alabama to Canada and from Maine and New Hampshire westward to Nebraska. The continuation of the rot during the storage period is common throughout this territory and probably ranks with blue mold (Penicillium) in frequency. Injury from the canker phase of the disease may be scarcely noticeable, or it may be so severe as to cause extensive killing or reduction in productiveness. In New York and adjacent territory great importance is attached to the canker phase of the disease, and the statement has been made that few orchards in that section are free from the disease. The extent to which the decline of trees from old age or from winter injury has played a part seems to have been largely overlooked, the presence of the black-rot organism being taken too often as the indication that it was responsible for the damage.

Etiology.—This disease is caused by one of the ascomycetous sphere fungi (*Physalospora malorum* (Berk.) Shear) which produces its ascigerous or perithecial stage in the bark of old cankers or on affected branches, and its pycnidial or *Sphæropsis* stage in the bark, in rotting fruit and more rarely in the leaf spots and on fallen leaves. The rarity of the pycnidial fruits in the leaf spots and the common occurrence of the pycnids of Phyllosticta or the fruits of other intruding fungi were for many years the source of misconceptions as to the true nature of the disease. The pycnidia are developed in abundance in the bark and on rotted fruits, but the perithecial stage seems to be rare, especially in America. The first report of the existence of a perithecial form of the black-rot pathogene was by Shear (1910 and 1914), who presented evidence that it was genetically connected with *Melanops quercuum* (Schw.) Rehm. forma *vitis* Sacc.

Arnaud, working in France (1912), described a fungus which he found on bark showing pycnidia of the black-rot type (*S. pseudodiplodia*) as *P. cydoniæ.* A year later (1913) Hesler found what he thought was the same fungus in America and proved by single ascospore cultures that it would produce typical pycnidia of *S. malorum*, and therefore accepted the finding of Arnaud. Because of the uncertainty of the identity of *P. cydoniæ* Arnaud as the ascus stage of *S. malorum*, Shear *et al.* (1924) have suggested *P. malorum* (Berk.) Shear as "a combination of the best-known names of the two stages of the fungus."

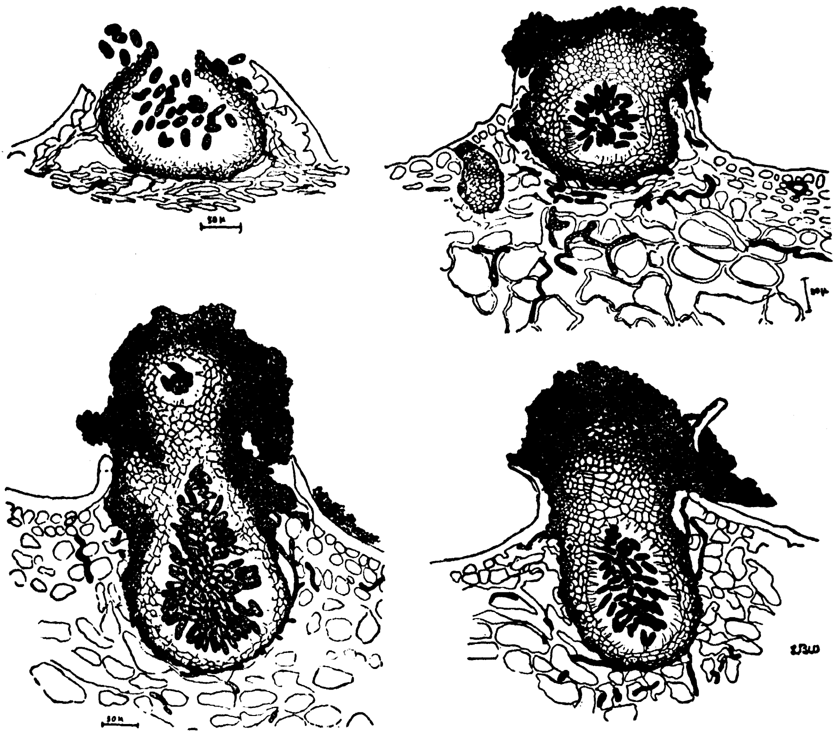

FIG. 184.—Sections through several types of pycnidia of the black-rot fungus. (*After Leva Walker, Neb. Rept.* **22**, 1908.)

The septate mycelium is intercellular and is at first hyaline, but soon darkens and becomes greenish yellow, bluish green, brown and then dark brown, appearing black in mass. Sclerotia-like bodies have been observed in cultures and beneath the skin of affected apples. Chlamydospores have been noted in cultures, as thick-walled, granular cells, occurring singly, in chains or in groups. Knots of hyphæ become aggregated just beneath the outer surface of the host parts and gradually develop into the pycnidia or conidial fruits.

The typical pycnidia are globose, subglobose, elongated or flask-shaped, black or carbonous, immersed in host tissues or becoming some-

what erumpent, ostiolate, and with distinct but variable outer and inner walls, the inner wall being of thin-walled, hyaline cells, while the outer is of dark, thick-walled cells. They are usually distinct but may be confluent or united into a stroma, a single pycnid measuring 200 to 300μ in diameter, while the compound structures may vary from 200 to 460μ high by 200 to 720μ wide. Hyaline, clavate or cylindrical conidiophores, 8 to 30μ long, arise from the inner face of the hyaline inner wall. Each conidiophore can develop a single, oblong-elliptical, brown spore, the sizes varying from 7 to 16.2μ broad by 16 to 36μ long. The pycnidia show wide variation as to form, size and detail of structure, and the pycnospores are likewise extremely variable. While the typical mature spore is brownish, they vary from nearly hyaline in the youngest mature spores to very dark brown in old, mature spores. The typical Sphæropsis spore is unicellular, but spores with one cross-septum are fairly common, and those with two or three are sometimes found. The hyaline color and continuous character are more common in young fruits, while the dark color and septation are more common in older fruits. This variation in color and septation is of special interest, since according to present classification of fungi the different conditions represent three different genera: the hyaline, continuous spores, *Macrophoma;* the colored, continuous spores, true *Sphæropsis;* and the one-septate, colored spores, *Diplodia.*

The perithecia have been found on twigs and cankers, and are very similar in form and size to the pycnidia. They are immersed in the cortical tissues and protrude by a short papillate ostiole. The wall is also similar in character to the wall of pycnidia, with inner hyaline cells and outer carbonous cells. Single fruits vary from 180 to 324μ high, by 300 to 400μ wide. Club-shaped asci, 21 to 32 by 130 to 180μ interspersed with hyaline, continuous paraphyses, arise from the base of the perithecium. The asci have thick walls at the tip, and contain from two to eight ascospores, eight being the common number. The ascospores are ellipsoidal or often inæquilateral, hyaline to greenish yellow, irregular biseriate in arrangement and vary from 10.8 to 15.2 by 23.4 to 34.2μ. The ascospores are forcibly expelled. "The wall of the ascus ruptures transversely and the ascospores are ejected while still embedded in a somewhat gelatinous matrix, having the same outline as the ascus and extending to the base where it appears to be attached" (Shear *et al.*, 1924).

The pathogene may be carried over the winter in the form of dormant mycelium, immature pycnidia, mature pycnidia, developing perithecia and possibly by pycnospores that have been set free and are lodged on the surface of the bark. Because of the rarity of the perithecia, it seems that these must play a very minor part in the life history of the pathogene, main reliance being placed on the pycnospores for the dissemination of the

fungus. The pycnospores accumulate in the pycnidial cavity, and are then pushed out through the ostiole, and may accumulate as short gelatinous tendrils, which will be washed away by rains due to the dissolving of the gelatinous matrix in which the spores are embedded. Rain and insects may bring about their further dissemination, but evidence of wind dissemination is lacking, since spores were not collected in spore traps set in orchards in which black rot was prevalent (Wolf, 1910). Their liberation in tendrils is also opposed to wind transport, and the great frequency of the pycnidia on dead twigs and the work of Walton (1920) showing that frog-eye infection is correlated with periods of rainfall make it unnecessary to assume any extensive wind dissemination.

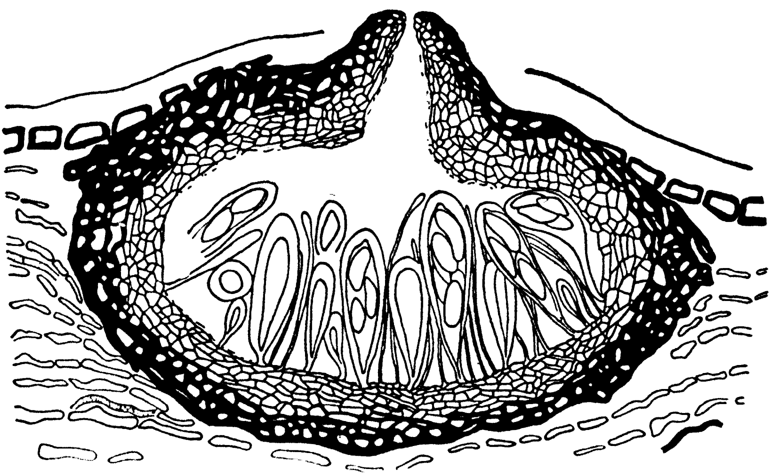

FIG. 185.—Section of a perithecium of *Physalospora malorum*. (*After Hesler, Cornell Univ. Agr. Exp. Sta. Bul.* **379**.)

The pycnospores are prevalent and ready to produce infections when conditions are favorable. Their retention of their vitality is an important feature in increasing the chances for infection. Hesler (1916) states that pycnospores that are 2 years old or older are still capable of germination, and there seems to be some evidence that their liberation is not confined entirely to the spring and summer, but may take place also during warm, moist periods at other times in the year. According to Preti (1926), the minimum temperature for germination is 9 to 10°C., the optimum 20°C. and the maximum 25 to 30°C. with light a necessary stimulus.

The time of infection of the foliage depends largely upon the condition of the host tissues. According to recent work:

Some frog-eye infection takes place during the blooming period, but most of it occurs from the time the petals fall until two and one-half weeks later, in other words when the leaves are in a growing condition (Walton, 1920).

It is certain that the pathogene can infect the uninjured leaf tissues and produce leaf spot. This behavior has been experimentally demonstrated by various workers, and, Swartout (1927) reports infection through sound, uninjured bark in greenhouse tests. In some localities fruit infection independent of injuries has been observed at the calyx end. Various types of injuries to the fruit or bark offer openings through which the fungus gains an entrance. These may be insect injuries by codling moth, curculio, etc. or other fungous or bacterial parasites, such as scab, blotch, bitter rot and fire blight may pave the way for entrance. Limb rub, hail bruises, spray injury, growth cracks, pruning wounds and frost cankers have been mentioned as furnishing infection courts.

Host Relations.—This disease is primarily a trouble of pomaceous fruits, being of first importance on the apple and of minor importance on the pear and quince. In addition, it is found either as a saprophyte or as a parasite on a considerable number of trees and shrubs, including "alder, apricot, ash, basswood, dogwood, elder, hawthorn, hop-hornbeam, lilac, maple, mulberry, oak, pine, rose, sumac, witch-hazel and others" (Hesler and Whetzel, 1917). On the quince it has been noted primarily as a fruit rot. The Sphæropsis on currant canes has been shown by Stevens (1924) to be the conidial stage of *Physalospora malorum* but distinct from the currant-cane blight fungus, *Botryosphœria ribis*.

Considerable variation has been noted in the susceptibility of varieties of the apple. The fruit rot is more severe on early varieties previous to maturity, while late or winter varieties are likely to suffer in storage. The leaf spot and frog eye are reported as severe on Ben Davis, Winesap, Arkansas, Baldwin and Jonathan in Virginia, on Ben Davis in Nebraska, on York Imperial and Stayman Winesap in Pennsylvania and on Chenango, Baldwin, Rhode Island and Twenty Ounce in New York. "In old orchards, particularly where pruning is neglected, the leaf-spot disease is much worse than in young orchards" (Scott and Rorer, 1908). Data on the susceptibility of varieties to the canker phase of the disease are mostly from New York and adjacent territory. In western New York Twenty Ounce is the variety most severely affected by canker. It is rarely found unaffected even in well-managed orchards, and neglected trees are often killed (Hesler, 1916). Paddock (1889) gives the following order of susceptibility of other varieties: Baldwin, Wagener, Rhode Island and Tompkins King. He says that Esopus has apparently run out because of its extreme susceptibility. Ben Davis and Northern Spy are severely affected in Ontario. Physiological strains have not been definitely established, but different isolations have been shown to vary greatly in rapidity of rot produced (Cooley and Fenner, 1926) and in behavior in cultures (Mohendra and Mitra, 1930).

Control.—As a basis for prevention or reduction in the amount of the disease the following features should be kept in mind: (1) that the

pathogene is a wound parasite on limbs and to a great extent on fruits; (2) the unbroken epidermis of leaves can be penetrated to produce leaf spot or frog eye; (3) cankers, blighted or dead twigs, fallen, spotted leaves and old mummies may all develop pycnidia which furnish spores; (4) the ascigerous fruits in old cankers may play a part in the spread of the pathogene.

Preventive or control measures may be grouped as follows: (1) prevention of wounds or their protection; (2) the treatment of cankers; (3) the removal and destruction of structures which furnish the inoculum; (4) spraying to protect the foliage or other parts. Many of the mechanical injuries may be avoided with a little care—for example, those due to machinery used in cultivating or spraying, by ladders and boots in pruning and picking, by props not carefully used or protected or by careless pruning or removal of water sprouts. Attention must be given to the control of insect pests and other fungous or bacterial diseases. Pruning wounds or other mechanical injuries that cannot be avoided should be treated with either a coating of coal tar or a Bordeaux paint. Attention should be given to cankers of whatever origin, since, if they are not already invaded by the black rot, they offer important courts of entrance. In all cases the grower must decide between the treatment of the canker and the sacrificing of the branch on which it occurs. The diseased tissue must be removed and destroyed and the wound protected or the cankered limb must be cut out. As a general principle for this or other diseases, cankers on large productive limbs or in the body of the tree should be treated, while small or unproductive cankered limbs should be removed. The destruction of black-rot mummies that are hanging on the tree or lying on the ground and very close pruning to remove all dead wood in which the fungus may be hibernating are commendable practices. All prunings and diseased bark, whether killed by black rot or from some other disease, should be destroyed by burning, since if left in the orchard they may continue to produce fruits of the pathogene. Either clean cultivation or the plowing under of the leaves previous to the blossoming period has been suggested.

The value of spraying for the prevention of canker is somewhat problematical. It would seem that limbs kept constantly covered with a Bordeaux should be afforded protection, but positive evidence of the value is not available. Spraying has been used effectively for the prevention of fruit rot, but more especially for the control of leaf spot or frog eye. Wolf (1913) has reported successful control in Alabama by the use of 4–4–50 Bordeaux about the middle of July followed by a second application 2 weeks later, and successful control was accomplished earlier by Waite (1901) and Scott (1905) in work on bitter rot. Commercial lime sulphur was reported entirely ineffective in controlling fruit rot due to Sphæropsis as well as to the bitter-rot pathogene.

Spraying was recommended for leaf spot before the connection with black rot was known. It is generally necessary to spray for scab in those regions in which Sphæropsis leaf spot is prevalent, and the usual experience has been that the disease is controlled by the scab sprays, the pink, calyx and one later spray generally being sufficient to secure practical control, while still further reduction will follow a fourth application, especially if the rains continue. The degree of control which may be expected may be judged from the following results:

Brooks and De Merritt (1912): Lime sulphur, 1–25, 26 per cent; checks 95 per cent.

Walton (1920): Lime sulphur, 1–30, 21 per cent; checks 79.4 per cent.

Walton (1920): Bordeaux, 4–5–50, 19.8 per cent; checks 79.4 per cent.

Crabill (1915) has reported 100 per cent efficiency in some cases, but it seems that perfect protection is rarely obtained. For leaf spot alone, self-boiled lime sulphur has given good results, but best control has been obtained from either commercial lime sulphur or Bordeaux. In Virginia, lime sulphur (Crabill, 1915) gave better control than Bordeaux, but in most cases the latter has been reported as slightly superior. On account of Bordeaux injury to both foliage and fruit, lime sulphur would seem more desirable unless bitter rot or blotch must also be controlled.

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CHESTNUT BLIGHT OR ENDOTHIA CANKER OF CHESTNUT

Endothia parasitica (Murr.) And. & And.

This is a virulent disease which invades the bark and cambium of twigs, branches or main trunk, forming cankers which ultimately girdle

these structures and cause the death of all distal parts, thus producing blight or dieback of twigs or branches, or leading to the death of the entire tree. The earlier reports of this disease have referred to it as the chestnut blight, the chestnut canker or the chestnut bark disease, but Endothia canker has been used more recently. Since a typical blight is not produced except by infections of small twigs, and other cankers of the chestnut are known, and since both bark and wood are affected during the progress of the disease, it might avoid confusion to adopt the specific name of *Endothia canker*, but "blight" is the term now most generally used by laymen, foresters and most plant pathologists.

History and Geographic Distribution.—This disease was first noted by Merkel in the New York Zoölogical Park in 1904, but was first described 2 years later (Merkel, 1906). The disease was studied by Murrill (1906) in the New York Botanical Garden and vicinity and was recognized as a serious trouble. The causal organism was described the same year as a new species, *Diaporthe parasitica.* The study of the disease was taken up in the U. S. Department of Agriculture very soon after the organization of the laboratory for the study of tree diseases in 1907, and the serious character of the trouble was realized. The disease spread with alarming rapidity and by 1908 (Hodson) was serious in portions of Long Island, Connecticut, Massachusetts, New York, New Jersey and Pennsylvania, radiating from the original infections in New York City and Long Island, and was reported from a number of outlying points in Delaware, Maryland and Virginia. The rapid march of the disease into Pennsylvania led to the establishment of the Pennsylvania Chestnut Tree Blight Commission in 1911 with an appropriation of $275,000 for the investigation and scientific study of the problem, and especially to ascertain the extent of the blight and to devise ways and means by which it could be stamped out. The investigations were also supported by a liberal appropriation by the Federal Department, and their investigations were conducted in cooperation with the Pennsylvania Commission until that went out of existence in 1913. The origin of the epiphytotic was one of the early questions for discussion, and two opposing views were held: (1) that the causal fungus was an obscure native of the United States that suddenly assumed prominence due to unfavorable conditions for its host—winter injury, and drought conditions (Clinton, 1909); (2) that the pathogene was an immigrant, introduced into this country from some foreign country, possibly Japan, with importations of nursery stock (Metcalf, 1908). This controversy was not settled until 1913, when the pathogene was discovered in China on native chestnuts (*Castanea mollissima*) by Meyer, an agricultural explorer of the U. S. Department of Agriculture (Fairchild, 1913; Shear and Stevens, 1913). In its native home the pathogene was not a serious parasite, making rather inconspicuous cankers, but when it reached America and found a very susceptible host in the native chestnut, *C. dentata,* it spread with alarming and deadly rapidity. The early study of the blight disease led to evidence that it had been prevalent some years previous to its discovery in New York and that probably more than one center of infection had existed. As a result of the increased interest in the disease and the financial support for investigations, the publications bearing on distribution, nature and control of the disease appeared in rapid succession in both the popular and scientific channels. Special impetus was given by the Pennsylvania Chestnut Blight Conference called by the governor in February, 1912, and in the course of a few years a voluminous literature had accumulated. A bibliography of the disease compiled to Jan. 1, 1914, included 399 titles (Beattie, 1914).

Experiments on control were attempted before the nature of the disease was understood, and eradication or control measures dominated the first efforts of the

Pennsylvania Chestnut Tree Blight Commission. Later attention was given to etiological and ecological relations, and important contributions to our knowledge were made by Anderson (1912–1914), Rankin (1912–1914), Heald and assistants (1913–1915) and others. The studies of the life history of the pathogene showed the futility of eradication measures after the disease had gained such headway, and as the best control practices had only served to retard the advance of the disease, funds were not provided for a continuation of the work and general control measures were largely discontinued. Since that time the disease has continued to extend its ravages. In 1914 the disease was generally prevalent in native chestnuts from New Hampshire and Warren County, New York, on the north to Albemarle County, Virginia, on the south and westward to a line running diagonally to the southwest through middle Pennsylvania. Scattering or spot infections occurred outside of this area in Maine, New York and Pennsylvania and at one locality in North Carolina. An orchard infection had been reported from British Columbia. Since that time the disease has continued its onward march to the south and west and has spread to nearly all parts of the range of the native chestnut and to some orchards beyond. It was discovered in Japan by Meyer in 1915 and reported by Shear (1916) as occurring on the native wild trees (*C. crenata*) which appeared to be even more resistant than the Chinese chestnut. It has recently been reported from Europe on the cultivated species (1924).

Symptoms and Effects.—Young infections of chestnut blight on smooth-barked vigorous shoots (2 to 6 or more years old) can be easily recognized by the presence of yellowish or yellowish-brown patches, slightly raised, and standing out in marked contrast to the olive-green healthy bark. The area invaded by the fungus may be fairly regular or very irregular in outline, the latter showing what has been designated as the amœboid type. In young infections of this type there are no fruiting pustules, but these make their appearance later. If the external brown layer of cork cells is removed from the advancing edge of the invaded area, the whitish or buff-colored mycelium, or vegetative body, of the blight fungus is exposed. Infections of this type may spread until the shoot is completely encircled, and fruiting pustules will be formed later.

Young infections on slow-growing twigs or on the smooth bark of older branches or trunks are not so evident, but they generally show as somewhat discolored, dead areas, sometimes slightly depressed and occasionally with a raised margin. The area invaded may be nearly circular, giving a so-called "target" infection, but it is more frequently elongated in the direction of the long axis of the shoot or branch. The invaded area gradually enlarges until the shoot or branch is completely encircled. A small shoot may be completely encircled before the appearance of fruiting pustules, but on larger limbs or on the main trunk the fruiting pustules begin to make their appearance long before complete girdling has taken place. These fruiting bodies show as small, yellow, orange or reddish-brown pustules (1⁄16 inch or slightly more in diameter) which break through the bark some distance back from the advancing edge of the lesion.

The interior tissue (inner bark invaded by the fungus) is changed to a yellowish-brown color, which is in marked contrast to the bright color

of the normal healthy tissue, and a careful examination by cutting away the bark will show the buff-colored fans of the fungus, which may have penetrated as deep as the cambium layer.

During damp weather following rains, or in moist situations, long, irregularly twisted threads, varying in color from buff to bright yellow, may be extruded from some of the pustules. These are masses of conidia or summer spores, and have been designated as "spore horns" or tendrils. The spore horns when first formed are soft and sticky, but when dry they become hard and brittle and are frequently darker in color.

Young infections on old trunks or large limbs with thick, fissured bark cause little change in the appearance of the bark itself and the fungus

FIG. 186.—Chestnut limbs showing cankers due to *Endothia parasitica.*

may have gained considerable headway before there is any external evidence of its presence. Sometimes the first indication of an infection on large limbs or trunks is the appearance of abnormal longitudinal splits or fissures. The orange or yellow fruiting pustules appear in the deep crevices or cracks, and spore horns may be developed from these under favorable conditions of moisture and temperature.

An infection with the blight fungus is sometimes the cause of a pronounced enlargement, or hypertrophy. This enlargement may involve the entire invaded portion or it may be more pronounced at the upper end of the lesion. Enlarged lesions are apparently the most frequent on vigorous shoots. Longitudinal splits or fissures in the bark are very characteristic of hypertrophied lesions. In many instances the lesion

may show a marked sunken area due to the killing of the invaded bark, while the surrounding tissues have continued to grow at the normal rate. This dead tissue may be more or less cracked or fissured and a typical canker developed. In the old lesions which have completely girdled a limb or branch the bark becomes cracked and fissured and begins to peel away. On old rough-barked trunks or branches the bark over old lesions will give a hollow sound when tapped, since the inner bark has been destroyed by the fungus. The bark may be readily peeled away and the inner fibrous portion is more or less shredded.

Aside from the discovery of the actual lesions there are various other symptoms which indicate the presence of blight: (1) dead leaves hanging

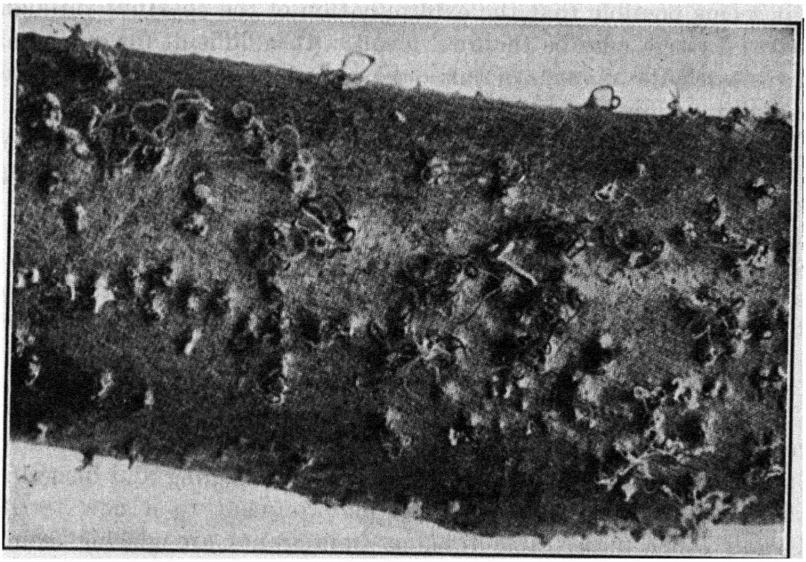

FIG. 187.—Piece of an infected branch showing numerous pycnidia and extruded spore coils or "spore horns."

in characteristic drooping clusters, which generally remain on the tree during the winter; (2) the persistence of burs, frequently undersized, on the blight-killed branches; (3) leaves of normal or reduced size become chlorotic, reddish brown or brown and fall, leaving defoliated limbs; (4) the development of groups of vigorous, rapid-growing sprouts at localized points on branches or trunk or at the crown, marking the location of a girdling lesion.

The final effect of the cankers is to kill the parts beyond the girdled zone, whether this is on twigs, branches or main trunk. The occurrence of trunk lesions is most serious, since, with the completion of the girdling, the entire tree must succumb. In trees which have suffered from top infections for several years, the occurrence of blight-killed branches gives a "stag-head" effect. The wood of blight-killed trees is injured but little as a direct result of the disease, but if left standing it soon begins

to deteriorate as a result of the work of insects and various species of wood-destroying fungi (Baxter and Gill, 1931).

Economic Importance.—The completeness of destruction wrought by the disease is without parallel in the annals of plant pathology. It has been making a clean sweep, killing young growth and merchantable timber of the forests, and the beautiful shade trees of cities and country estates throughout the invaded territory. The chestnut tree has a wonderful capacity to sprout from stumps or the base of killed trees, but these young vigorous sprouts are soon killed by the disease, and finally no further growth is possible. In the areas of earlier infestations the fine chestnut forests, groves and shade trees have entirely disappeared, and it seems possible that the extermination of the chestnut throughout its natural range may be the final result. It is difficult for one who has not witnessed the ravages of this disease to appreciate the thoroughness of the destruction.

During the earlier years of the epiphytotic various attempts were made to estimate the financial losses from the disease. According to Metcalf (1913):

> The estimate of $25,000,000 made in 1911 as representing the loss up to that time was probably much too conservative. But the total loss to date is insignificant compared with the loss which will ensue if the disease once attacks the fine chestnut timber of the South Appalachians.

The disease has continued unchecked since that time and the losses have amounted to more than that caused by any other forest-tree disease. All this destruction has happened because a struggling and insignificant parasite was carried from its home environment to a new territory. This and other undesirable fungous immigrants are glaring examples of the early failure of this country to establish and maintain an efficient inspection of imported stock.

Etiology.—This chestnut disease is due to *Endothia parasitica* (Murr.) A. and A., one of the sphere fungi or Sphæriales, which produces a Cytospora-like pycnidial stage and Valsa-like stromata containing the perithecia. This fungus was first referred to the genus Diaporthe by Murrill, but the studies of Farlow, Clinton, Shear and the Anderson brothers (1912) have shown that it is an Endothia and distinct from the native American species, which are saprophytic. Rehm (1907) referred this parasite to the Hypocreales (ergot fungi and allies) because of the bright-colored stromata, and named it *Valsonectria parasitica* (Murr.) Rehm, but this binomial was never accepted by American pathologists.

Endothia parasitica has been shown by repeated inoculations to be true to its specific name, successful inoculations being readily made by pure cultures, or by mycelial transfers from active lesions. It is, however, strictly a wound parasite, being unable to penetrate the unbroken

periderm or bark of either young or old parts. The mycelium develops principally in the living cambium and bark and to a more limited extent in the outer sapwood of any part of the host above ground, and continues to grow in dead parts as long as moisture and food are available. The mycelium is at first a cottony white, but soon becomes buff or yellowish and spreads out in the bark layers or in the cambium in the form of closely appressed sheets or fan-like layers.

A month or more after an infection the mycelium begins the organization of the fruiting pustules for the production of pycnospores. These *pycnidial pustules* or *stromata* appear as minute raised papillæ scarcely larger than a pinhead, and show a yellowish or orange color when they break through the bark. Each pycnid is a dense aggregate of fungous tissue, generally containing one (rarely more) large, lobulated cavity, lined with innumerable vertical filaments or conidiophores, by which

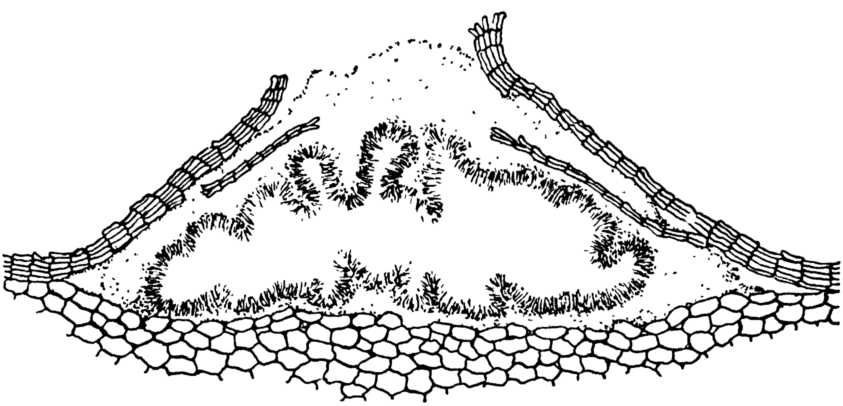

FIG. 188.—Section through a single pycnidium.

enormous numbers of minute rod-shaped bodies, the *pycnospores*, are produced. These pycnospores are hyaline, continuous, cylindrical, straight or slightly curved with rounded ends and average 1.28 by 3.56μ. Due to their accumulation in the pycnidial cavity the external wall is ruptured and the spore mass embedded in mucilaginous material oozes out in the form of a yellowish or orange, thread-like or flattened, irregular coil, the so-called "spore horn" or tendril. These spore horns are produced during moist periods following rains, and persist until washed away during some later storm. During rainy periods the pycnospores are being formed and washed away from mature pycnidia as fast as they are forced out to the surface. A single spore horn of average size has been found by actual analysis to contain as many as 115,000,000 pycnospores.

After a period of activity the pycnidial pustules may be transformed into perithecial stromata, which then show upon their surface a number of raised papillæ or a number of minute black dots, the ostioles or openings of the perithecia or flask-like bodies buried deep in the stromata.

Each perithecial stroma contains one to 60 (average 15 to 30) distinct flask-like perithecia, each of which opens to the surface by a long, black neck. The body of the perithecium is 350 to 400μ in diameter, the outer

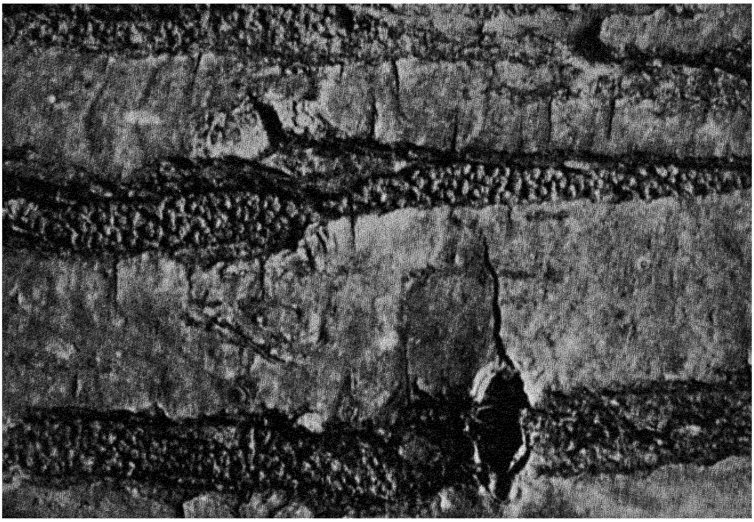

FIG. 189.—Perithecial stromata in the crevices of rough bark

FIG. 190.—Groups of perithecial stromata somewhat enlarged.

wall made up of 10 or 12 layers of compact, dark, heavy-walled cells, the inner wall of two or three layers of thin-walled cells from which the asci originate. The neck of the perithecium is lined with thin-walled

hyphæ which project inward and upward and are especially prominent near the ostiole. The asci are oblong or broadly clavate, eight-spored, average 8.9 by 51.2μ and have a very delicate hyaline wall with a thickened ring at the apical end. The ascospores are irregular uniseriate or subbiseriate in arrangement, hyaline, oblong to oval, one-septate, generally constricted at the septum and average 4.5 by 8.6μ. In mature perithecia, when sufficiently moist and temperatures are favorable, the asci are detached from the wall, and as they accumulate are forced up the long neck in a linear series. When an ascus reaches the ostiole and

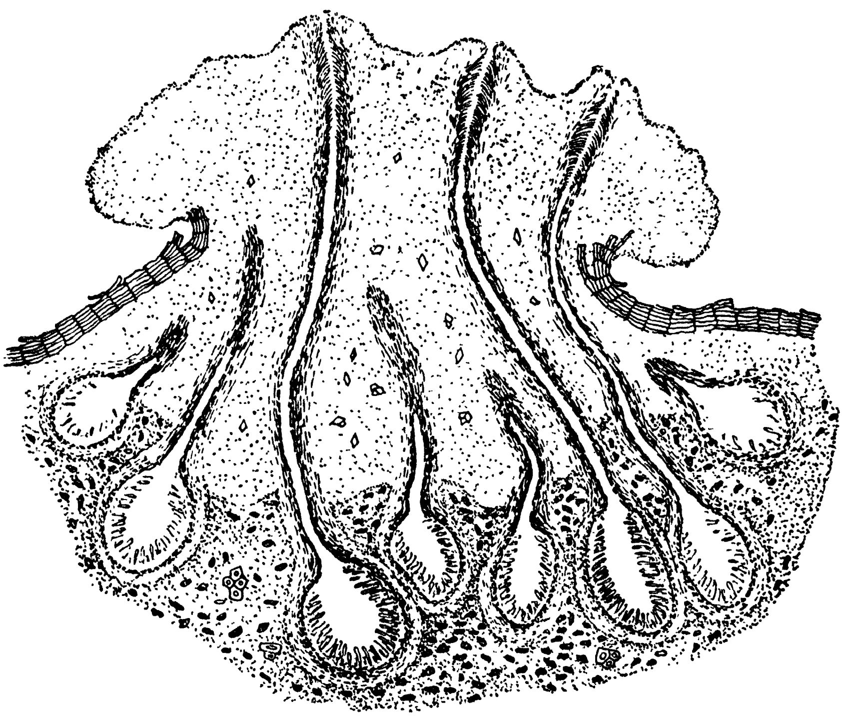

FIG. 191.—Section of a perithecial stroma showing immersed, flask-shaped perithecia which open to the surface by long necks.

its tip is exposed to the exterior, it explodes and the eight ascospores are projected into the air. Another ascus is pushed up to take its place, and in due time it expels its charge of spores. This process continues as long as new asci can be formed and conditions are favorable, so each perithecium may be called a repeating spore gun, which is repeatedly firing its load of spores into the surrounding air. It must be evident that these ascospores, by their method of discharge, are adapted to a wind dissemination, while the pycnospores, being embedded in a mucilaginous matrix readily soluble in water, are more suited to dissemination by rains.

The pycnospores have frequently been designated as "summer spores," but the development of the pycnidia depends largely upon the age of the lesion rather than on the time or season of the year. Pycnospores are produced in abundance at all times of the year when temperature and moisture conditions are favorable, and are washed down in large numbers from diseased branches even during the warm winter rains, when the spore horns are rarely observed. The ascospores have been designated as the "winter spores," but their time of maturing also depends

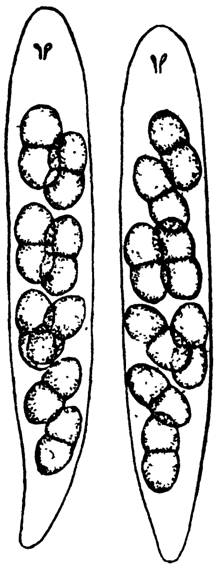

FIG. 192.—Asci showing form and position of ascospores.

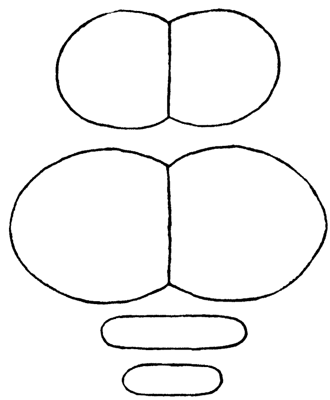

FIG. 193.—A comparison of maximum and minimum sizes of ascospores and pycnospores.

more upon the age of the lesion than upon the season of the year. Maturing perithecia may be found at any season of the year, although they are perhaps more abundant in the fall and winter than at other seasons. Successive crops of perithecial stromata may be found on a single lesion which has persisted for a number of years. The pathogene may spread throughout the bark of a blight-killed tree and continue to produce fruiting pustules, or they may be formed in abundance in the bark of fallen logs. Pycnids may even be formed on the bare wood of peeled logs or on the cut ends of the sapwood or on fragments of bark or wood left in chopping. The chestnut-blight fungus has a remarkable power of spore production, both pycnospores and ascospores, and either form seems equally effective in producing infections.

The pycnospores have been shown to survive for 2 to 3 months in a perfectly dry soil; they have been found in viable condition on normal bark below lesions as long as 14 days after a rain; they are washed down

from lesions with every winter rain in enormous numbers, but are even more numerous in the spring and summer; they are carried in large numbers on the bodies of insects which frequent cankers, as many as 336,000 being obtained from a single insect (*Leptostylus macula*); they are also carried by birds which frequent chestnut trees in search of insects, analyses showing that a single downy woodpecker had over 750,000 that were still viable on its feet, bill and head. Although early statements were made that the pycnospores were wind disseminated, no evidence of such spread has been obtained. Rain, insects and birds are the important agents of their dissemination and, considering their resistance to desiccation, they might even be transported long distances on the bark of nursery trees or in soil adhering to their roots, even though the stock was itself free from infection.

The drying of bark does not prevent the perithecia from expelling ascospores when again subjected to favorable moisture and temperature conditions. When dried in the laboratory for 2 months, spore expulsion was active after 2 days' exposure to moisture, and activity was even resumed by stromata that had been dried for nearly a year.

The expulsion of ascospores takes place only under natural conditions when the stromata have been moistened by rains but before they have dried. Tests have shown ascospores to be very prevalent in the vicinity of infected chestnuts during the first 5 hours following the cessation of rains, and less abundant later, in some cases being prevalent as long as 14 hours after the rain. During winter rains the temperature is too low for expulsion to take place. At 38.6°F. no spores were expelled, only a few at 53.8°F., while the optimum temperature for expulsion was 68 to 80°F. (Heald and Walton, 1914). It is interesting to note that this optimum for spore expulsion agrees very well for the optimum for growth as given by Shear and Stevens (1913). "Perithecia show an almost phenomenal power of spore production, as shown by the fact that spores were expelled from some specimens every day for 168 days. Some perithecia were still active when the test was discontinued" (Heald and Walton, 1914).

Expulsion of spores from the perithecia of *Endothia parasitica* begins in the spring with the first warm rains and increases to a maximum of activity as conditions become more favorable, to be followed by a decline in the fall when lower temperatures prevail, and ceases entirely during the cooler portions of the year, although there may be an abundant rainfall. During one-third to one-half of the year there is then no expulsion with each rain of any consequence (Heald and Studhalter, 1915).

Activity of pustules is not exhausted in a single season, but may be as vigorous during the second season as during the first. This remarkable power of ascospore production is due to successive maturing of asci, successive maturing of perithecia and to successive maturing of stromata throughout the season.

It has been shown conclusively that the ascospores are wind-disseminated. By exposure plates and spore traps it has been shown that ascospores are carried away from diseased trees in large numbers following each warm rain of any amount. They have been obtained in large numbers from the air 300 to 400 feet from the source of supply, hence the conclusion is justified that they may be carried much greater distances. It is of interest to note that the period of their prevalence coincides with periods of moisture when conditions should be favorable for infections.

All attempts to make infections with either spores or mycelium through the normal bark have been failures. It was thought that the lenticels might offer an avenue of entrance, but there is no evidence that such is the case. Injuries of some sort which open the bark are necessary. Such injuries may be made in a variety of ways, but it is believed that certain insects are the most important agents in making openings. A bast miner which tunnels in the bark and emerges to pupate in the soil is very abundant throughout the natural range of the chestnut, and infections can frequently be found which appear to center at the exit openings of this insect.

After an infection has been established the mycelium continues to grow, its rate depending mainly on the temperatures which prevail, rains having little or no effect on its advance. Anderson (1914) gives the average annual rate of growth or increase in the diameter of the lesion around the tree, based on a large number of measurements at Charter Oak, Pa., as 15.97 centimeters. The growth was the most rapid during the summer months, but continued during the warmer periods of the winter months. The annual rate of growth has been shown to be much more rapid at the southern range of the disease (Charlottesville, Va.) than at the most northern locality (Concord, N. H.), where it was only about half that at the southern point. At a given latitude the rate of growth also decreases with the altitude. These data would indicate a more rapid spread of the chestnut blight in its southern advance (Stevens, 1917).

Host Relations.—All varieties of chestnuts, including the native American species, *Castanea dentata*, and the European and Japanese species, are susceptible to the Endothia canker. The eastern chinquapin (*C. pumila*) and the western chinquapin (Castanopsis) can also contract the disease, but show considerable resistance. As previously noted, the Chinese and Japanese species show a decided resistance, so much so that in their native country they suffer but little injury from the disease. It is unfortunate that the native American species, the most valuable species commercially, should be so exceedingly susceptible. The breeding problem in relation to the Endothia canker consists of producing resistant varieties for nut production and resistant varieties that will be profitable timber trees. Considerable progress has already been made in the production of resistant horticultural varieties by hybridizing (Van Fleet,

1914) and the introduction of resistant species and strains from the Orient (Galloway, 1926).

Endothia parasitica can grow as a saprophyte on the bark of various trees and has been found growing naturally in the woods on several species of oaks (Quercus), *Acer rubrum*, *Carya ovata* and *Rhus typhina*. Inoculations on various species established the fact that the fungus was only very weakly parasitic on *Quercus alba* and *Q. prinus*, and led to the conclusion that the canker fungus is not a menace to other forest trees except the chestnut.

Control.—The control of the Endothia canker has been attempted under strictly forest conditions, in nut-producing orchards and in ornamental shade trees, but with little success in any case. Early in the epiphytotic careful pruning and tree surgery or the cutting out of cankers alone or combined with spraying were used in the attempt to save valuable shade and orchard trees, but despite the most painstaking efforts owners saw their prized trees gradually go down. The most thorough practice of this method of treatment only served to prolong the life of the affected trees, since they were constantly reinfected. This method of treatment for individual trees would probably be successful in regions outside of the chestnut belt, where trees would not be exposed to infection from the outside. Internal therapeutics or injections of toxic chemicals into the bark resulted in neither cure nor protection (Rumbold, 1920).

The possibility of limiting the state or nation-wide spread of the disease through the forests was given special attention by Federal and state authorities. While most foresters and pathologists believed that it would be impossible, Pennsylvania was not willing to give up the fight without making a special effort. The plan finally tried was first to locate spot infections in the region beyond the area of general prevalence of the disease, and then an attempt was made to eradicate these spot infections by cutting out and destroying the infected trees, following methods which were planned to reduce the chances of reappearance of the disease. Even the most careful handling of these spot infections was not completely effective. Of 42 spots in which eradication was attempted, 14 showed a recurrence of the disease. What result would have followed a second eradication was not determined, since Pennsylvania failed to provide funds for the continuation of the work, which was discontinued in July, 1913. Since that time only sporadic efforts have been made by state authorities in any of the affected states and the disease has continued its forward march.

Within the invaded forest areas the problem has become one of the utilization of the chestnut timber in the most efficient way, since control has been abandoned. Blight-killed timber deteriorates quite rapidly, hence should be cut and marketed as soon as possible (Gravatt and Gill, 1930), and the devastated areas devoted to other desirable species.

Studies on natural replacement in the hardwood forests of the Northeast have shown stands running very largely to oaks (Korstian and Stickel, 1927).

The only hope for the chestnut is the breeding of resistant or immune varieties. The extermination of the American chestnut throughout its range seems certain unless nature herself intervenes. Extermination of species of trees has characterized former geologic times. It may be that such a process is being enacted before our eyes at the present time.

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IMPORTANT DISEASES DUE TO SPHERE FUNGI AND ALLIES

I. HYPOCREALES

Hypocreaceæ.

Cankers of apple and pear and other woody species (*Nectria galligena* Bres. *et al.*).—CAYLEY, D. M.: Some observations on the life history of *Nectria galligena* Bres. *Ann. Bot.* **35**: 79–92. 1921. ZELLER, S. M.: Cankers of apple and pear in Oregon and their control. *Ore. Agr. Exp. Sta. Circ.* **73**: 1–29. 1926. ——: European canker of pomaceous fruit trees. *Ore. Agr. Exp. Sta. Bul.* **222**: 1–52. 1926. WOLLENWEBER, H. W.: Nectria-Krebs. *In* Sorauer's Handbuch der Pflanzenkrankheiten **2**: 550–558. 1928. MORITZ, O.: Studien über Nectriakrebs. I. Infektionsversuche. *Zeitschr. Pflanzenkr.* **40**: 251–261. 1930.

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False smut of rice and maize (*Ustilaginoidea virens* (Cke.) Tak.).—HASKELL, R. J. AND DIEHL, W. W.: False smut of maize, Ustilaginoidea. *Phytopath.* **19**: 589–592. 1929.

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II. DOTHIDIALES

Dothidiaceæ.

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Black knot of currant and gooseberry (*Plowrightia ribesia* (Pers.) Sacc.).—HOGAN, I. A.: The parasitism of *Plowrightia ribesia* on the currant. *Trans. Brit. Myc. Soc.* **12**: 27–44. 1927.

III. SPHÆRIALES

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Bark fungus of stone fruits (*Calosphæria princeps* Tul.).—Although this fungus occurs on bark of living trees as well as on dead branches it does not seem to cause material injury.

Black rot, canker and leaf spot of apple (*Physalospora malorum* (Berk.) Shear).—(See special treatment, p. 629.)

Blight of stone fruits (*Ascospora beijerinckii* Vuill.).—The ascigerous stage of this fungus has not been found in America. (See Blight of Stone Fruits, under Imperfect Fungi, p. 707.)

Dieback of peach, plum and other stone fruits (*Valsa leucostoma* Fr.).—ROLFS, F. M.: Winter killing of twigs, cankers and sun scald of peach trees. *Mo. Fruit Exp. Sta. Bul.* **17**: 9–101. 1910. WORMALD, H.: The Cytospora disease of the cherry. *Jour. S. E. Agr. College, Wye* **1912**: 367–380. 1914. LEONIAN, L. H.: The physiology of perithecial and pycnidial formation in *Valsa leucostoma*. *Phytopath.* **13**: 257–272. 1923. TOGASHI, K.: Morphological studies of *Leucostoma leucostoma and Valsa japonica*, the causal fungi of canker or dieback disease of peach trees. *Bul. Imp. Coll. Agr. and For. Morioka, Japan* **14**: 1–50. 1930. ——: Comparative studies on the physiology of *Leucostoma leucostoma* and *Valsa japonica*. *Ibid.* **15**: 1–76. 1930.

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CHAPTER XXIII

DISEASES DUE TO IMPERFECT FUNGI

FUNGI IMPERFECTI

In the great groups of fungi many species produce at least two types of spores or spore fruits in the course of their life cycle, as follows:

	I	II
Black molds and allies (Zygomycetes)...	Zygospores	Sporangiospores or conidiospores
Downy mildews and allies (Oömycetes)..	Oöspores	Conidia or swarm spores
Powdery mildews and allies (Perisporiales)......................	Perithecia	Conidia of the Oidium type
Sphæriales and allies (Pyrenomycetes)...	Perithecia	Conidia of various types
Cup fungi and allies (Discomycetes).....	Apothecia	Conidia of various types
Smut fungi (Ustilaginales)............	Chlamydospores	Conidia rarely
Rust fungi (Uredinales)...............	Telia	Uredinia and æcia
Palisade fungi and allies (Hymenomycetes)............................	Basidium fruits	Conidia of various types

Perfect and Imperfect Stages.—The spores or spore fruits indicated in the first column make it possible to assign a fungus to a definite family or order in the great groups of fungi and to determine the genus and species, and are considered the *perfect stages.* The spore forms or spore fruits indicated in the second column represent the *imperfect stages.* These are sufficiently characteristic in some groups to show positively the perfect stage with which they are connected, but in many forms the imperfect stages alone offer no certain clue to the perfect stages to which they are related. In such cases the uncertain forms are kept together for convenience as the *Fungi Imperfecti,* which represent a heterogeneous group of very diverse forms. The group may then be considered as a temporary resting place for forms the affinities of which are not known or cannot be safely predicted. It is probable that certain fungi have lost their power to produce the perfect stage, while in a great number of cases the perfect form exists but the relationships have not been discovered. Many of our important plant pathogenes which were formerly known only from their imperfect stages have during the last 20 years been connected with their proper ascus or basidial stages.

Classification.—Three large subdivisions of the imperfect fungi are recognized as follows:

I. Hyphomycetales or Moniliales.—The conidia are produced upon an undifferentiated mycelium or on specialized conidiophores, either single, fascicled or grouped into extensive layers or united to form coremia or sporodochia. Four groups with the rank of families are recognized:

1. *Moniliaceæ.*—Conidia from an undifferentiated mycelium, or on specialized conidiophores, single, fascicled or grouped into extensive layers. Mycelium and conidia generally clear or hyaline.
2. *Dematiaceæ.*—Same as Moniliaceæ, except hyphæ and conidia typically dark.
3. *Stilbaceæ.*—Spore fruit typically a coremium.
4. *Tuberculariaceæ.*—Spore fruit typically a sporodochium. In the Stilbaceæ and Tuberculariaceæ some species may produce the fruits characteristic of the Moniliaceæ or Dematiaceæ when developed on certain substrata.

II. Melanconiales.—Spore fruit typically an *acervulus.* On certain media the Hyphomycetous type may be assumed.

III. Sphæropsidales.—Spore fruit a pycnidium. Four groups with the rank of families are recognized:

1. *Sphæropsidaceæ.*—Pycnidia of varying shapes, globose, conic or lenticular, and membranous, carbonous or coriaceous, *black.*
2. *Nectrioidaceæ.*—Pycnidia as in the Sphæroidaceæ, but fleshy or waxy and *light colored.*
2. *Leptostromataceæ.*—Pycnidia irregular or shield-shaped, more or less dimidiate and *black.*
4. *Excipulaceæ.*—Pycnidia cup-shaped, patellate or hysteroid, more or less globose at first, but finally widely open and *black.*

The most important genera of the Moniliaceæ, Dematiaceæ, Melanconiales and Sphæropsidaceæ, together with the spore types, are indicated in the accompanying tabulation.

In addition to the genera listed in the accompanying tabulation, a few genera of other families contain important plant pathogenes. The following are of special interest:

Stilbaceæ:

Spores hyaline, single-celled: Stilbella, Coremium, Isaria.
Spores dark, single-celled: Stysanus.
Spores dark, one- to several-septate: Isariopsis.

Tuberculariaceæ:

Spores hyaline, single-celled: Sphacelia, Tubercularia, Tuberculina, Volutella, Illosporium.
Spores dark, single-celled: Strumella.
Spores hyaline, macroconidia one- to several-septate and straight or curved; microconidia smaller and generally non-septate: Fusarium.
Spores dark, several septate, straight or curved: Exosporium, Trimmatostroma.

Leptostromataceæ:

Spores hyaline, single-celled: Glœodes, Leptostroma, Leptothyrium, Melasmia.
Spores hyaline, four-celled in the form of a cross, each cell with a delicate appendage: Entomosporium.
Spores linear or filiform, continuous or septate: Brunchorstia, Leptostromella.

Excipulaceæ:

Spores hyaline, one-celled: Amerosporium, Dothichiza, Sporonema.

Mycelia-sterilia:

No spore stage known: Rhizoctonia, Sclerotium, Ozonium.

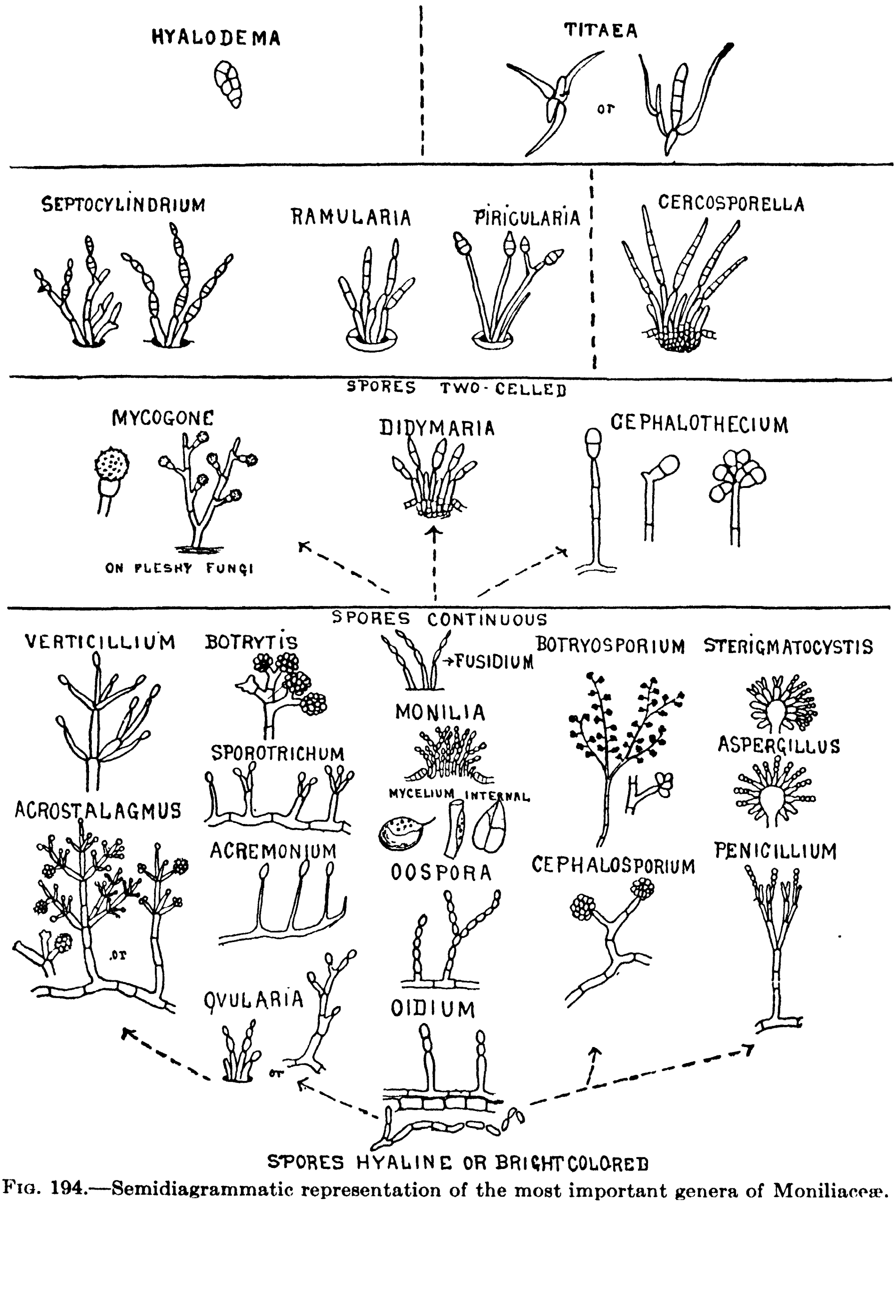

FIG. 194.—Semidiagrammatic representation of the most important genera of Moniliaceæ.

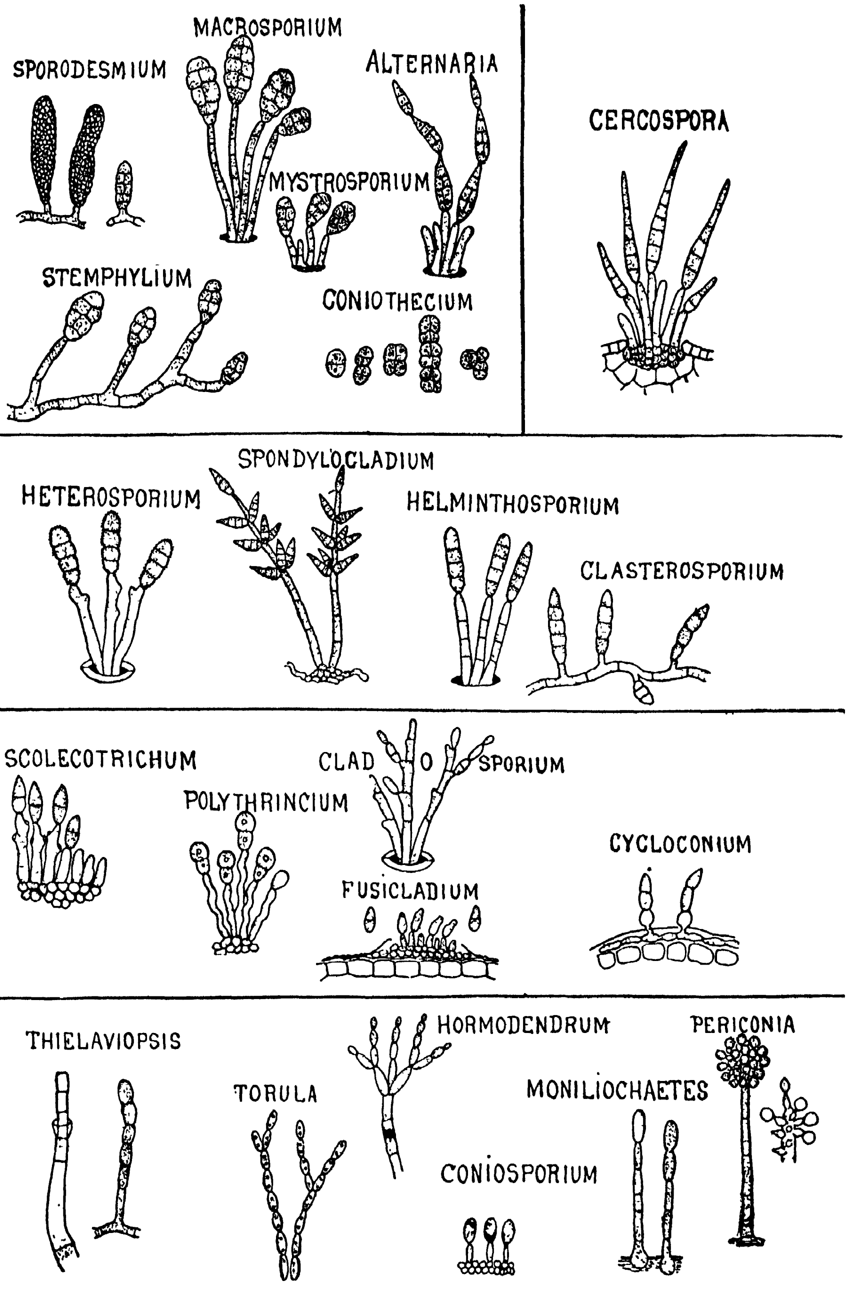

FIG. 195.—Semidiagrammatic representation of the most important genera of Dematiaceæ.

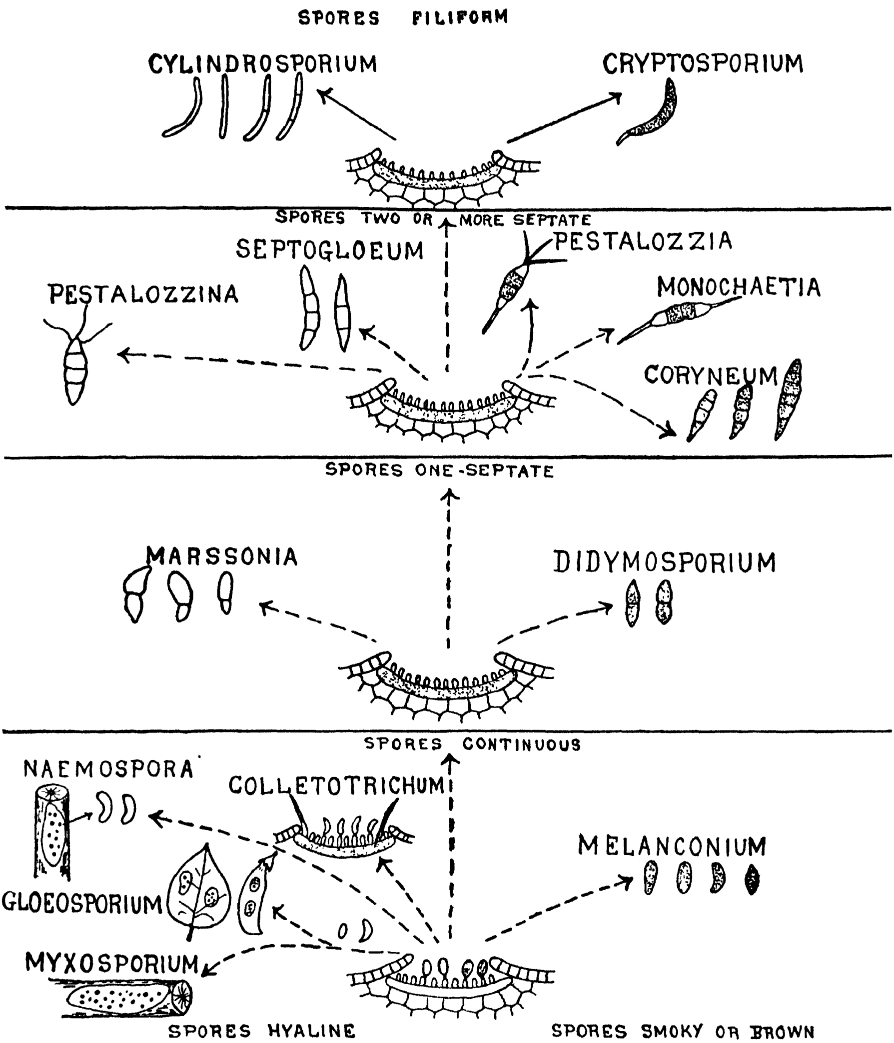

FIG. 196.—Semidiagrammatic representation of the most important genera of Melanconiales.

Type of spore fruit	Conidiophores single, fascicled or grouped into extensive layers	
Type of spore	Moniliaceæ, hyaline spores	Dematiaceæ, dark spores
Muriform	Hyalodema	Macrosporium Alternaria Sporodesmium Mystrosporium Stemphylium Coniothecium
Elongate-fusoid, clavate or filiform, continuous or one- to several-septate	Cercosporella	Cercospora
Ovoid to cylindric, two- to many-septate.	Septocylindrium Ramularia Piricularia	Heterosporium Spondylocladium Ceratophorum Helminthosporium Stigmina Clasterosporium
Two-celled or one-septate	Mycogone Didymaria Cylindrocladium Cephalothecium	Scolecotrichum Polythrincium Cycloconium Cladosporium Fusicladium
Single-celled or continuous	Sterigmatocystis Aspergillus Penicillium Botryosporium Cephalosporium Trichoderma Verticillium Acrostalagmus Pellicularia Botrytis Sporotrichum Acremonium Ovularia Fusidium Monilia Oöspora Oidium	Coniosporium Moniliochætes Periconia Hormodendrum Torula Thielaviopsis

Acervuli	Pycnidia	Pycnidia
Melanconiales	Sphæropsidaceæ, hyaline spores	Sphæropsidaceæ, dark spores
	Hyalothyris	Camarosporium
Cryptosporium **Dark spores** Cylindrosporium **Hyaline spores**	Rhabdospora Phleospora Trichoseptoria Septoria	Phæoseptoria
Monochætia Coryneum Pestalozzia **Dark spores**		
Pestalozzina Septoglœum **Hyaline spores**	Kellermania Stagonospora	Cryptostictis Hendersonia
Didymosporium **Dark spores** Marssonina **Hyaline spores**	Darluca Diplodina Ascochyta	Chætodiplodia Botryodiplodia Macrodiplodia Diplodia Microdiplodia
Melanconium **Dark spores** Næmospora Colletotrichum Glœosporium Myxosporium **Hyaline spores**	Dothiorella Cytospora Fusicoccum Cytosporella Ampelomyces Vermicularia Plenodomus Phomopsis Macrophoma Phoma Phyllosticta	Sphæropsis Coniothyrium

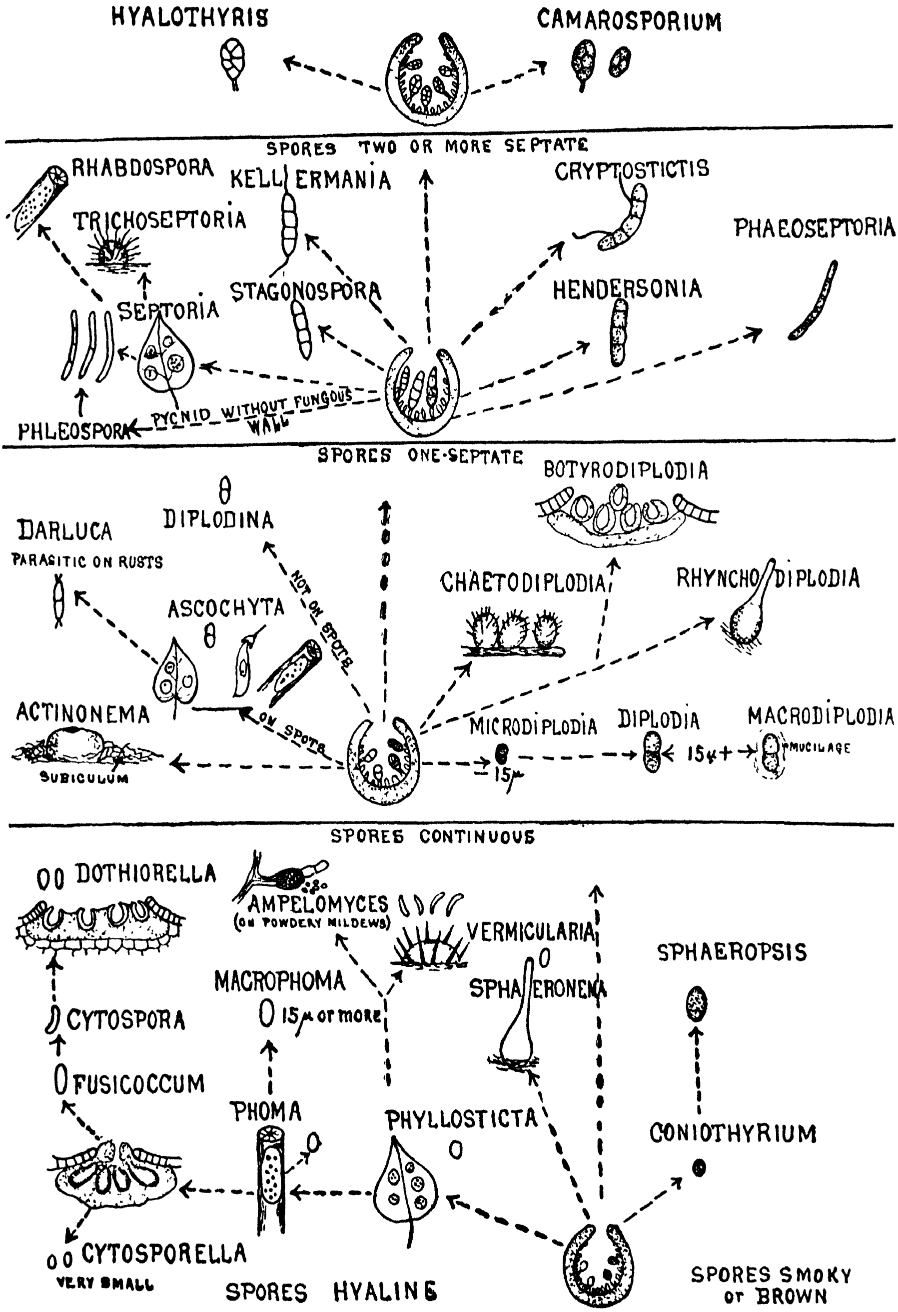

FIG. 197.—Semidiagrammatic representation of the most important genera of Sphæropsidaceæ.

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EARLY BLIGHT OF POTATO

Alternaria solani (E. & M.) Jones & Grout

This is a disease characterized by target-board spotting of the foliage with a resultant blight. While it has generally been designated as early blight to distinguish it from the late blight or Irish blight (*Phytophthora infestans*), it has been suggested that leaf spot is a more expressive name (McAlpine). It is also called leaf curl in Australia. In order to avoid confusion with other foliage troubles of the potato, the name Alternaria blight may be used to distinguish it from the Phytophthora blight.

History and Geographic Distribution.—Although the causal fungus had been described some years previous, the disease was first reported to be of economic importance by Galloway (1891), but a few years later (1893) complaints of its serious character came to the U. S. Department of Agriculture from various parts of the country. The disease was studied by Jones in Vermont (1893), Chester in Delaware (1893) and by Sturgis in Connecticut (1894). Later studies by Jones (1895–1896) led him to the conclusion that the trouble to which he had given the name of early blight, was a true parasitic disease. The disease was noted by Sorauer in Germany in 1896 and he referred the causal organism to Alternaria, to which conclusion Jones had arrived about the same time. It was noted by McAlpine in Australia in 1896. These early reports of the disease from widely separated parts of the world confirm the idea which has been expressed that the disease had long been prevalent but had not been distinguished from late blight and other foliage disorders. During following years the prevalence of the disease was recorded from various portions of the United States and foreign countries. But little was added to our knowledge of the etiology of the disease until the work of Rands (1917).

Early blight has been recorded from Mexico to Canada in North America, from all the other continents, and from Java, Bermuda, Australia and New Zealand.

"Thus it probably occurs wherever the potato is an important crop. As to whether the parasite is native to the potato and has spread with it from its original home in South America to the various countries into which the potato has been introduced is largely a matter of speculation" (Rands, 1917).

Symptoms and Effects.—The disease is characterized by the appearance of dark-brown or almost black, more or less circular dead areas upon the leaflets, which usually show a concentric series of rings or ridges giving the lesions a "target-board" effect. The young spots are small and pale in color, and become darker and more irregular in form with their increase in size. Under certain conditions the spots may remain small and more or less angular, being limited by some of the smaller veins, and in such cases the target-board effect is indistinct. A spot may show a marginal faded zone as the trouble advances, but the affected area remains

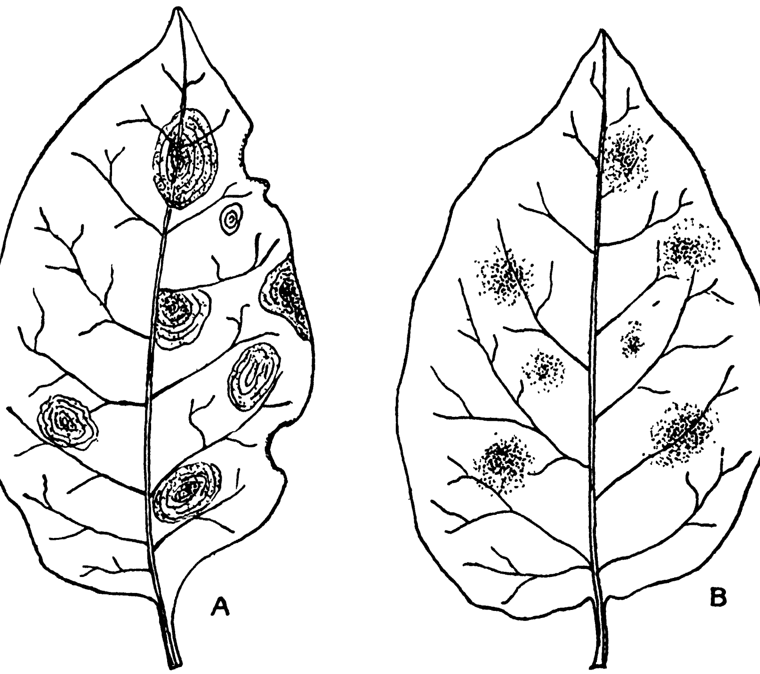

FIG. 198.—*A*, potato leaflet showing leaf spots due to *Alternaria solani*; *B*, leaf spots due to *Cercospora concors*.

sharply defined. The spots may be few in number and small or they may be numerous and involve a large part of the leaf area. Adjacent spots may coalesce to form more extended dead areas, and with progress of the disease the leaves may show ragged margins or irregular breaks, or sometimes perforations due to the brittleness of the dead tissue. In severe cases the final result may be a complete blighting of the affected leaves, frequently preceded by the yellowing of the tissue between lesions. Affected leaves may also show more or less curling or rolling. The stalks of seriously affected plants may turn yellow and dry up, and brown lesions may also appear on these. The various leaf troubles of the potato may be readily separated by the following characters:

1. Lesions marginal or terminal and spreading to involve extended areas or the entire leaf. Whitish mold-like growth on the under surface during damp weather—*Phytophthora blight.*

2. Diffuse leaf spots, without sharply defined borders and lacking any concentric zonation—*Cercospora spot.*

3. Margins of leaflets blighted, curled up and more or less broken—*tip burn and hopper burn.*

Arsenical poisoning, sun scald and necrosis of leaf tissue which results from degeneration diseases, may also cause leaf spotting or blighting.

The lower leaves are generally first affected, followed by the upper and younger leaves in severe attacks, but the disease does not reach its maximum until the plants have passed their period of greatest vegetative vigor. In the late stages of the trouble most of the lower leaves may be blighted, leaving only a few green spotted leaves at the top of the plant.

Whetzel (1923) has recently reported an epiphytotic of early blight in Bermuda, in which the symptoms and effects were somewhat different from those commonly seen. The onset of the disease was sudden and its progress rapid, the devastation was extreme, the leaf lesions were large and quite similar to those of late blight and large water-soaked lesions appeared on the stems.

The effect on the crop may be considerable, as the attack often coincides with the period when the developing tubers throw a strain on the nutrient resources of the plant. The tubers are not directly attacked and are never rotted, but they remain small, immature, soft skinned, difficult to keep and deficient in starch (Butler, 1918).

More recently Folsom and Bonde (1925) have described a rot of the tubers due to the early-blight organism and suggest that the infection may result from the contact of freshly dug tubers with diseased foliage. The importance of this phase of the disease has more recently been emphasized (Gratz and Bonde, 1927; Pittman, 1929). Tubers showing no evidence of the disease when harvested may develop severe storage rot when shipped to southern markets. It is difficult, however, to determine the actual loss from early blight, since insect injuries and other diseases are generally an accompaniment. The disease has been more severe in the eastern and southern United States than in other countries, with the possible exception of Australia, New Zealand and South Africa. McAlpine considered early blight a minor trouble in Australia and Butler wrote that there was no indication that it would become a dangerous pest in India. While it is present in the United States west of the Rocky Mountains, it is relatively rare and seldom is sufficiently prevalent to call for special control measures.

Jones (1903) states that in certain seasons *Alternaria solani* causes more loss in many parts of New England than does the mildew (late blight). Several cases

are on record of unusual attacks, but more important, however, is the smaller but yearly toll of the disease. Coons (1914) averages the annual loss in Michigan as about 25 per cent. In Wisconsin Jones (1912) states that it may reduce the yield 10 to 25 per cent (Rands, 1917).

Etiology.—Early blight is caused by *Alternaria solani* (E. & M.) Jones & Grout, one of the imperfect fungi belonging to the Dematiaceæ

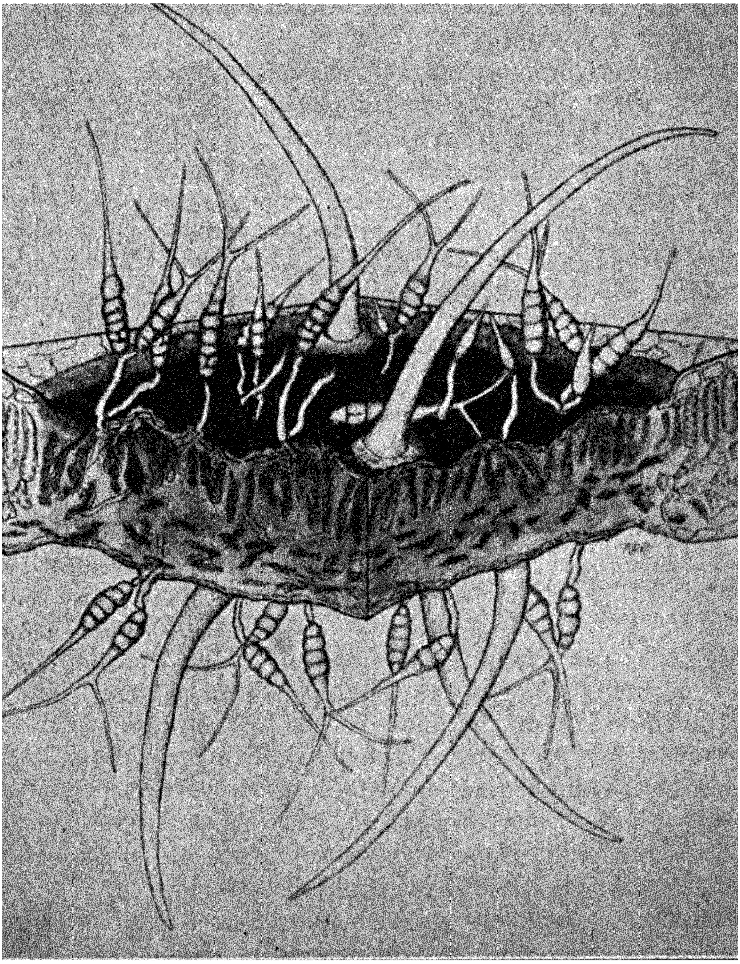

FIG. 199.—Diagrammatic representation of an early-blight spot showing effect on tissues and the production of spores. (*After Rands, Wis. Agr. Exp. Sta. Res. Bul.* **42.**)

of the Hyphomycetes. Jones first proved that the fungus is a real parasite and can attack perfectly healthy tissue. It was first described as a *Macrosporium* by Ellis and Martin in 1882, but was assigned to Alternaria because of the discovery by Jones (1896) and Sorauer (1896) that the spores were sometimes formed in chains when the fungus was grown in

cultures. Catenulate spores have never been found on the leaf lesions, and for this reason Duggar (1909) and others have objected to calling the pathogene Alternaria. Lindau (1923) still retains the original name, and established usage seems to be the only reason for retaining the name Alternaria.

The fungus produces a light-brown or olivaceous, septate mycelium ramifying in the intercellular spaces and also penetrating cells within the diseased tissues. After the invaded tissue has been killed, brown, septate, erect or ascending conidiophores, 50 to 90 by 8 to 9μ, emerge from the stomata or push out to the surface (either upper or lower) between the dead cells. Each conidiophore gives rise to a single spore, this arising as a bud or outgrowth from the terminal cell. This bud is at first a minute hyaline projection which grows rapidly and enlarges to form the mature spore, and this is easily separated from its point of attachment. The spores or conidia are "obclavate, brown, terminating in a very long hyaline septate beak (frequently branched) equaling fully one-half the length of the spore." The body of the spore shows 5 to 10 cross-septa, but longitudinal septa are either few or lacking. The spore size has been given as 145 to 370 by 16 to 18μ, but more recent measurements by Rands are 120 to 296 by 12 to 20μ. If the spores are washed off from a spot a new crop will be produced under favorable conditions, as many as three or four successive crops being recorded. The conidia are able to germinate at once under favorable conditions of moisture and temperature. Five to ten germ tubes may arise from the different cells of a spore, or the number may be reduced to two or three if the temperature is rather low. Infection may take place through stomata or directly through the epidermal walls or insect injuries made by flea beetles, or Colorado beetles may serve as avenues of entrance.

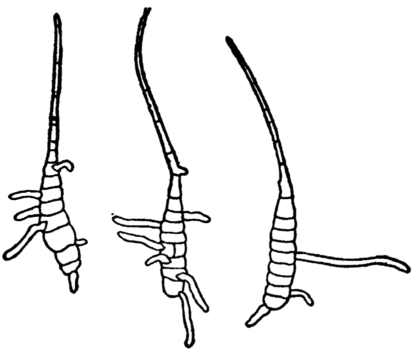

FIG. 200.—Germination of spores of *Alternaria solani*. (*After Rands, Wis. Agr. Exp. Sta. Res. Bul.* **42**.)

No perfect or ascigerous stage has ever been found, either on overwintered leaves or in cultures. The source of the inoculum for the first infections in an early crop is supposed to be either the over-wintered spores or new spores developed from mycelium that has persisted in the soil or in the plant remains from a former crop. Massee, who reports that the disease is carried over from one season to another by a dormant mycelium in the tubers, must either have been considering another trouble or had a very imperfect knowledge of the life history of the pathogene. That conidia and mycelium suffice to carry over the fungus is evidenced by the

results cited by Rands that dried diseased leaves yielded the pathogene in cultures after 12 and 18 months, and that conidia showed a 10 per cent germination after 17 months at room temperature. Early infections may produce spores to be carried to later maturing plants, wind and insects being the principal agents of dissemination. Under favorable conditions the period of incubation is relatively short, incipient spots showing 48 to 72 hours after inoculation while spore production occurs within three or four days. The fungus shows a marked tendency to saltate, and physiological strains are recognized (Bonde, 1929; Pittman, 1929) varying in cultural characters and in pathogenicity.

Early blight ordinarily makes little development until the host has passed its period of greatest vigor and is being weakened by external conditions or by the drains of tuber formation. Optimum spore production is dependent upon frequent rains aided by heavy dews. Climate and soil exert a controlling influence upon the development of the spores. In general, it becomes most serious when the season begins with abundant moisture, which is followed by high temperatures unfavorable to the host plant but with sufficient moisture to insure maximum sporulation. Periods of continued drought check its spread completely. The conclusion is, therefore, reached that the optimum conditions for an epidemic of early blight require relatively high temperatures alternating with moist periods in combination with a more or less weakened condition of the plant (Rands, 1917).

Host Relations.—The pathogene of early blight can also infect tomatoes and eggplants, and the appearance of the spots on these hosts is similar to that on the potato. It has also been found as the cause of severe rotting of tomato fruits shipped from the South to northern markets, and is reported from Wisconsin as causing a severe blighting of eggplants in the seed bed. Early blight is especially important as a tomato disease in Louisiana, where, according to Edgerton and Moreland (1913), it is nearly as serious as "wilt," and may cause losses of 50 per cent. In the North the disease is frequently found on tomatoes, but it is rarely serious. *Alternaria solani* will make incipient infections which never enlarge and become spore-producing on a considerable number of species of Solanum and related genera, while on others typical sporulating lesions are formed. Rands (1917) records six species of Solanum besides potato, eggplant and tomato as hosts. These include the common black nightshade and the garden wonderberry (*S. nigrum guinense* L.). White henbane (*Hyoscyamus albus* L.), black henbane (*H. niger* L.) and apple of Peru (*Nicandra physaloides*) are also susceptible. The leaf spot on Jimson weed (Datura), which has been attributed to the same fungus, has been shown by Rands to be due to a distinct species which he recognizes as *A. crassa* (Sacc.) Rand. It should also be mentioned that another species, *A. fasciculata*, which produces an abundance of spores in chains on natural substrata, is common as a saprophyte on blight lesions on the potato and various other species of the nightshade family.

Variations in the susceptibility of varieties have been noted, but most of those showing resistance have been potatoes of poor commercial qualities. The McCormick variety reported as very resistant by Norton has been tested by Rands and proved the most resistant of 15 varieties with which it was compared. It also is of poor quality so it is of main value as a promising parent for the breeding of new resistant varieties.

Control.—As the basis for a rational control it should be noted: (1) that the pathogene lives over winter in the débris from preceding crops; (2) that the disease is primarily a foliage trouble, resulting from purely localized infections which originate from wind- or insect-borne spores; (3) that tuber infections originate from spores produced on diseased tops. Crop rotation will at once be suggested as of importance, and in case of continuous cropping all dead vines should be raked together and burned immediately after harvest, but these practices must be regarded only as aids to the control by spraying.

It has repeatedly been demonstrated that both early and late blight can be controlled by spraying with Bordeaux (see Late Blight). In most localities both diseases are present, but in Rhodesia, where early blight is the important disease, increases in yield of 16 to 57 per cent have been reported from the use of Bordeaux (Jack, 1913, 1916), and in Georgia, where the late blight is also important, increased yields have resulted from the use of Bordeaux (Stuckey and Higgins). It should be recognized that there are many localities in which early blight is not sufficiently frequent or serious to justify the expense of spraying, hence local experience as to the prevalence of the disease must be the guide in determining the number of applications of fungicide or whether spraying may be omitted.

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BEAN ANTHRACNOSE

Colletotrichum lindemuthianum (Sacc. & Magn.) Bri. & Cav.

This is a disease of the common bean (*Phaseolus vulgaris* L.) which affects seed, seedlings, leaves and other vegetative parts, but makes its typical and characteristic development upon the pods. It has been called bean "spot disease," "speck," "pod canker," "pod spot" and "rust," but the name "anthracnose," which was first used for the disease by Scribner (1888), is now generally employed. This name is derived from a Greek word meaning ulcer, and is appropriate because of the ulcer-like lesions on the pods. While gardeners frequently use the name "rust" for the disease, it should be understood that this is an incorrect use of the word, which should be reserved to apply only to the diseases caused by true rust fungi. Bean blight is an entirely different disease due to a bacterial pathogene (*Pseudomonas phaseoli* E. F. Smith).

History.—The disease was first discovered by Lindemuth at Bonn, Germany, in 1875 and was first described a few years later by Saccardo (1878), although it is known that it occurred previous to that time in France and other parts of Europe. The first detailed work on the disease was carried out by Frank (1883), who studied it in Germany and determined its infectious character and also established the important fact that it may be seed-borne. It was recognized in the United States previous to the report of Scribner (1885) and attention was soon given to the disease by Halsted

in New Jersey (1891–1901), Beach in New York (1892) and again in New York by Whetzel (1906 and 1908). The importance of the disease in Louisiana is emphasized by the appearance of several bulletins by Edgerton (1909–1916). The study of anthracnose in New York was continued by Barrus (1911), and after 10 years of detailed and exhaustive studies he published his work and a résumé of that done by previous investigators (1921). The disease was sufficiently serious to call forth two bulletins by Muncie (1914 and 1917) from the Michigan Experiment Station. Mention should also be made of the recent work of Fischer in Germany (1919); of Dey in England, on the method of penetration of the infection hyphæ; of McRostie (1919, 1921), Ten Doornkaat-Koolman (1927) and Reddick (1928) on the inheritance of anthracnose resistance; of Leach in Minnesota (1923), Burkholder (1923), Muller (1926) and Budde (1928) on biologic forms. Many other shorter articles have appeared and the disease has been frequently reported in the agricultural literature of this and foreign countries. In 1921 Barrus cited 170 publications dealing specifically or remotely with anthracnose. The monographic treatise of Schaffnit and Böning (1925) and the resistance tests of Rands and Brotherton (1925) are the most recent important contributions.

Geographic Distribution.—Anthracnose is world wide in its distribution, having been reported in either moderate or severe form from the various countries of Europe, from Japan and the Asiatic continent, from South Africa, Australia and New Zealand and from North and South America. In North America the disease has been reported from the extreme south to as far north as Alaska, but it is not a factor of commercial consequence in all parts of the United States. It seems to reach its greatest severity in the states east of the Rocky Mountains, and is frequently the cause of heavy losses from the Dakotas and Nebraska eastward to the Atlantic Coast. The prevalence in the South is evidenced by the statement of Edgerton (1909) that it is the greatest drawback to the growing of beans in Louisiana. The scarcity of the disease west of the Rocky Mountains is evidenced by the practice of eastern seedsmen in obtaining western-grown seed because of its relative freedom from infection. The rarity of the disease is responsible for the development of the bean seed-growing industry in Colorado and Idaho in recent years. Horne reports in 1921: "Very rare and unimportant in California." Although eastern-grown seed has repeatedly been planted in Washington, no field development of the disease has been brought to the attention of the writer in 16 years' experience. In visits to the large Seattle markets the writer has never been able to locate even traces of anthracnose. From these statements it appears that the West, particularly the Pacific Northwest, enjoys an unusual freedom from this serious disease of the eastern and southern United States.

Symptoms.—The anthracnose affects directly all parts of the host except the roots (rarely) from the seedling stage to mature plants, but it is especially noticeable and characteristic on the pods. The first evidence on these is the appearance of minute brown or rust-colored spots in the epidermis, generally obliquely oriented with reference to the axis of the pod. They rapidly enlarge, the center becomes sunken and darker in color, almost black, while a rusty-brown, hazel-colored or even reddish border persists around the outside.

As the pod matures, the lesion is marked at the edge of a canker by a slightly raised, black ring with a cinnamon-rufous to chestnut-colored border. The center of the spot is then somewhat light buff in color. Flesh-colored spore masses on the surface of a young canker dry down to gray, brown or even black granulations or to small pimples (Barrus, 1921).

The lesions are generally more or less circular in outline, and range in size from mere specks to spots 1 centimeter or more in diameter. When cankers are close together or numerous there may be more or less fusion of spots and irregular or extended lesions may result. The spots are typi-

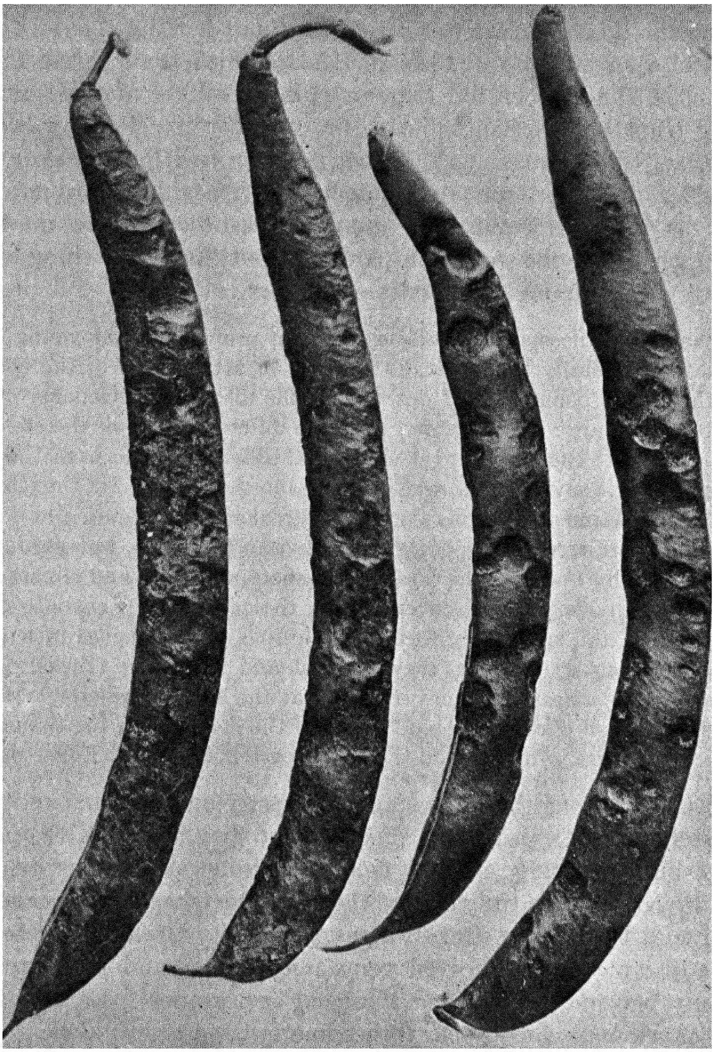

Fig. 201.—Anthracnose on pods of beans. Pod at the left completely covered with anthracnose lesions.

cally sunken due to the drying up and collapse of the cells in the center, and, with the presence of the pink, sticky spore masses that develop during moist weather, have quite an ulcer-like appearance. Some lesions may be confined to the wall of the pod, while others penetrate the entire thickness of the endocarp and then involve the underlying seed. Early

infections are more liable to penetrate into the seed, while later infections may remain more superficial.

A number of other troubles cause pod lesions, which should be distinguished from those caused by anthracnose: (1) blight lesions (*Pseudomonas phaseoli*), irregular and more extended water-soaked spots, extending along the dorsal suture or on the sides, but lacking the dark color or the pinkish spore masses; (2) lesions due to Rhizoctonia, quite similar to anthracnose, but generally larger, without spore masses, and only affecting pods lying in contact with the soil; (3) invasions by *Sclerotinia sclerotiorum*, which cause a soft, brown, moldy rot with internal or external black sclerotia; (4) true rust infections (*Uromyces appendiculatus*), evident as pustules containing dusty, dark-brown spore masses. In addition, there may be some non-parasitic spotting or blotching of pods if they have been exposed to intense sunlight.

On the stems the spots sometimes develop profusely. On young seedlings the spots are generally below or at the point of attachment of the cotyledons, due to the spores being washed down from the diseased cotyledons. On older plants, the spots are more scattered over the different parts of the stems. The spots appear quite suddenly on the stems and differ but little from those on the pods. However, they are, as a rule, elongated in the direction of the main axis of the stem. Sometimes, also, there are dark streaks extending up and down from the spots. These streaks are quite distinct near the spots, but gradually fade out in the healthy tissue of the host. The spots are black and slightly sunken, and, as on the pods, become covered with the pink, slimy exudate of spores. By coalescence, the spots may form large lesions 3 or 4 inches in length. As these large lesions grow older, they dry out and the tissue cracks. Often on young plants the disease becomes so bad that the stem completely rots and we have a damping-off effect. On older parts of the stem, when the tissue becomes hard, the spots rarely form; when they do they remain small (Edgerton, 1910).

In leaf infections the lesions appear mostly on the veins and petioles. The latter may be so seriously affected that they are not able to support the leaf blade. Under normal field conditions the lesions on the leaf blades are confined to the veins of the lower surface, which become black in color and later show sunken areas, especially the larger veins. Leaf tissue adjacent to the infected veins may wither and turn brown, and later may become torn, giving a ragged appearance. Early attacks on young leaves may cause them to become twisted or crinkled.

The other parts of older plants may become diseased, and lesions similar to those already described may appear on the branches of the plant and on the pedicels, the sepals and the bracts of the inflorescence. These parts of plants grown out of doors, however, become affected only during protracted periods of weather favorable to the organism (Barrus, 1921).

The disease appears in the seed as yellowish, brownish or blackish spots, frequently quite evident on the white-skinned varieties, but

generally very obscure on the dark-skinned varieties. The spots vary in size from mere specks to lesions involving half of the bean, and may or may not be sunken. The lesions frequently extend through the seed coat and involve the underlying cotyledons. In the young seedlings the black cotyledon lesions enlarge, become more depressed and develop sticky spore masses the same as the pod lesions.

Effects and Economic Importance.—Bean anthracnose causes losses due to (1) reduced viability of infected seed; (2) poor stands due to death of affected seedlings; (3) reduced yields due to direct pod infections or to retarded growth; and (4) poor quality of the harvested crop, either snap or string beans or the threshed product. Infected seed may fail to germinate or the young seedlings may be killed before they emerge from the soil or soon after, with the result that poor stands are obtained. The disfigured pods are unsalable as string beans, and this effect on quality is emphasized by the fact that "southern-grown beans, apparently healthy when shipped, frequently reach northern markets in a badly spotted condition, and if the disease is common in the field, pods kept over night after picking are likely to be rusted the next morning" (Barrus, 1921). Dry beans from severely affected fields will be of poor quality because of the shrunken and spotted seed, and will suffer a certain percentage of dockage on the market to compensate for the inferior quality.

While bean anthracnose has taken a certain annual toll since its appearance, its prevalence in many sections has been marked by periods of epiphytotic intensity in which enormous losses have been experienced. In the eastern United States there were epiphytotics during 1906–1908, from 1914–1915 and also in 1917. The loss in New York in 1915 was estimated as $700,000, while for the same year in Michigan a loss of $3,000,000 has been reported (Muncie, 1917). In Michigan between 1913 and 1916, the growers and bean jobbers reported an average pick per bushel of 6 pounds, about 50 per cent of which consisted of anthracnose-spotted seed. Cases of individual fields showing losses of 100 per cent have been reported. It is believed that the continued planting of affected seed was one of the important factors in the production of epiphytotics, and that with care in seed selection epiphytotics will be less frequent.

Etiology.—This disease is caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav., one of the imperfect fungi, belonging to the Melanconiales. The pathogene was first named *Glœosporium lindemuthianum* by Saccardo (1878), but setæ were discovered in the acervuli by Scribner (1888) and the following year Briosi and Cavara proposed the present binomial. The earlier name is still used by some German writers (Lindau, 1923), perhaps with some propriety, since it has been shown that the presence or the absence of setæ is not a constant character, and therefore not sufficient for the determination of generic position. In 1913 Shear and Wood reported that they had produced perithecia

belonging to *Glomerella* in cultures obtained from bean pods, but the connection of these with the anthracnose fungus was never proved and it is doubted if there is any ascus stage. At least none of the other numerous workers have ever obtained perithecia either in nature or in cultures.

The mycelium of the pathogene is localized in the tissue of a lesion and does not spread internally to other parts. After it makes a certain development it organizes the fruiting bodies, or *acervuli*, below the epidermis, in the center of the lesions. Each fruit consists of a stromatic layer from the surface of which are formed simple, erect, hyaline, continuous conidiophores, 45 to 55μ in length, packed closely together. Conidia are produced at the tips of the conidiophores and with their

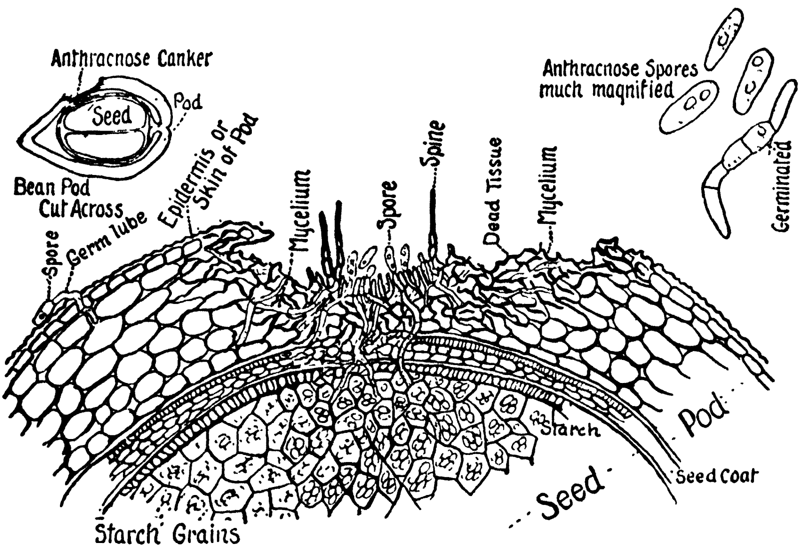

FIG. 202.—Semidiagrammatic section of pod lesion due to *Colletotrichum lindemuthianum*, showing relation of the anthracnose fungus to the tissues of the bean. (*After Whetzel, Cornell Bul.* **255**, 1908.)

accumulation the epidermis is ruptured, while these conidia, held together by a mucilaginous secretion, form the pink, slimy masses which appear on lesions. In some cases stiff, pointed, unbranched, septate, brown hairs or setæ, 30 to 90μ long, arise between the conidiophores or around the margin of the acervulus. These setæ are more abundant in older fruits, and Krüger (1913) has been able to develop setose and non-setose cultures by varying conditions. The conidia have an average size of 15 by 5μ but vary from 13 by 4.44 to 22 by 5.33μ, are hyaline (pink *en masse*), continuous, oval or oblong, straight or slightly curved and are produced in enormous numbers. A single lesion may bear a few to 50 or more acervuli and these may continue to form conidia for a considerable period, producing new crops after the old spores have been washed away

by rains. Edgerton estimated that as many as five hundred million to one billion spores may be produced on a single pod.

Since the conidia are held together by a gelatinous coating, they are not wind-disseminated, but during rain periods they are separated from each other and washed away. Wind-blown rain or spattering rain drops or moisture on the surface of affected structures may become filled with a suspension of spores, which may thus reach new host parts. On germination on a susceptible host a conidium produces a short germ tube, which on coming in contact with the epidermis enlarges at the end and forms a brown, thick-walled, more or less angular cell, the *appressorium.* This becomes closely attached to the surface by its mucilaginous envelope, and is apparently a device to facilitate infection, since the appressorium which is cemented to the cuticle soon produces a "peg-like infection hypha" which grows out from its under surface and "ruptures mechanically the cuticular layer, and then brings about swelling of the subcuticular layers, no doubt by enzymic action" (Dey, 1919). The infection hypha after growing a short distance into the host, either within the subcuticular layers or within the cell invaded, produces a small enlargement or vesicle, from which branches originate that spread through the tissue. Once within the cells the hyphæ continue to advance (except in resistant varieties) and a typical lesion soon results. Under favorable conditions the first evidence of infection can be detected in 24 to 36 hours and typical sporulating lesions may be formed in 4½ to 9 days, the variation being due largely to environmental factors and the susceptibility of the host.

One of the most important features of the disease is the penetration of the pathogene into the seed, where it may pass the winter as a dormant mycelium, either within the cells of the seed coat or, in badly diseased seeds, within the cells of the cotyledons or as spores between the cotyledons or between the seed coats and embryo. The mycelium may sometimes be found as a cobweb-like growth between the seed coats and the cotyledons or between the cotyledons, and spores may be formed either on acervuli or without the organization of such fruiting bodies. Whatever the condition of the fungus in the seed, it resumes activity and soon becomes spore-producing when germination of the seed begins. The infested seeds are thus the most important sources of the first infections which appear in the fields.

The first infection of young seedlings has been described as follows:

The plumule, consisting of the young leaves, is, during germination, and for a short time thereafter, in intimate contact with the cotyledons. By capillarity and in other ways the water containing the suspended spores comes in contact with the now exposed under surface of the leaves. The stem may also become inoculated with sporeś that have been washed down from the cankers on the coty-

ledons, especially the base of the stem just below the surface soil, where moisture conditions are favorable for the germination of the spores (Barrus, 1921).

Infections may also take place from spores washed down into the soil by rains, either before the emergence of the seedlings or while they are still very young. After the initial infections new sources of spores become available and the disease will spread from plant to plant through the field. Spores may be disseminated by splashing rain drops, by contaminated drops of water that may be carried by the wind to healthy plant parts, by wet leaves being blown against one another, by the dripping of water from diseased parts to healthy structures—in fact, by any process which transfers moisture from one part of the plant to another. Even the agitation of the vines by pickers soon after a rain or when wet with dew may serve to carry the spores to new locations and thus spread the disease.

The question has been raised as to the longevity of the free conidia, and tests have shown that they are not viable to any extent after 7 weeks. This would mean that disseminated conidia cannot carry the fungus over the winter, when mingled with the soil or if lodged on the surface of healthy seed. The behavior of the fungus on the old vines and pods is of special concern, for it seems that the fungus might be carried over on these in inactive fruits or by a dormant mycelium. Trials made by Muncie (1917) and by Barrus (1921) and some earlier workers have led to the conclusion that "the fungus is capable of living from one season to another, and even for two seasons, in old affected vines and pods, which may serve as a source of inoculation when carried to the field." While observations have shown that beans planted in fields which had produced a previous diseased crop became more severely affected than the previous crop, and that fertilizing with bean-straw manure has increased the amount of the disease, the opinion prevails that infections from such sources are of minor consequence.

Conditions Favoring Anthracnose.—The prevalence or severity of this disease is influenced by the temperature, moisture in the soil and air, rain and dew. The optimum temperature for the growth of the fungus ranges from 71.6 to 73.4°F. and the maximum has been given at 86 to 93°F. The range of temperature at which infections will result has been given by Lauritzen (1920) as 57 to 80°F. In the South, according to Edgerton, the moisture and the temperature conditions which prevail in the early part of the growing season are favorable to infection and spread of the disease, but from June to August it is absent from the fields even with ample rainfall and when diseased seed is used, since the temperatures are close to or beyond the maximum endured by the pathogene. The temperature is undoubtedly the most important factor in excluding the disease from the Pacific Northwest, but here it is probably the low night temperature which prevents infection. These low night temperatures, coupled with scanty rainfall, meager dews and an abundance of sunshine,

make it practically impossible for the anthracnose to develop even though diseased seed is planted. In the eastern United States the average climatic conditions are generally favorable for anthracnose, but the occurrence of epiphytotics can be explained largely by the increased rainfall through a period of years. In New York the disease reached a maximum in 1906. The 3 or 4 years previous to this date had been increasingly rainy and there had been a gradual increase in the severity of the disease. The season of 1906 was a very rainy one, especially when the beans were young and when the pods were forming, and the gradual increase in the disease in previous years had produced seed heavily infected, so that the two factors favored the unusual severity of the epiphytotic. Late planting to produce a fall crop in Louisiana has given practically a disease-free crop, while in New York it is claimed that "later maturing beans are the most liable to damage from anthracnose, as weather conditions favorable for infection commonly occur during September" (Barrus, 1921).

It may be noted that conditions which permit the retention of moisture beneath the vines are particularly favorable for infection of the pods. For this reason a dense stand in moist soil promotes the disease, and drill planting is more favorable for it than check rows.

Host Relations.—Anthracnose is primarily a disease of various varieties of common beans (*Phaseolus vulgaris* L.), but it has been reported as affecting a few other species of the genus. Scarlet Runner and White Dutch Runner (*P. multiflorus* Willd.) are only slightly susceptible, and the same is true of Lima beans (*P. lunatus* L.), although both dwarf or bush and pole varieties have been successfully infected. The tepary bean (*P. acutifolius latifolius*) has been severely infected when inoculated in the seedling stage, and slight infections have been obtained on *P. aureus* Roxb. At various times different workers have reported the occurrence of anthracnose on the cowpea (*Vigna sinensis* Endl.), common peas (*Pisum sativum* L.), horse bean (*Vicia faba* L.) and jack beans (*Dolichos spp.*). In regard to these occurrences Barrus writes as follows:

> There are evidently but few species outside the genus Phaseolus which are susceptible in any degree to anthracnose, and no plants except varieties of *Phaseolus vulgaris* are susceptible to such an extent that the disease becomes epiphytotic in regions where such plants are extensively grown. It is not unlikely that several strains of the pathogene exist, one capable of infecting *P. multiflorus* or some of its varieties in a severe manner, another able to attack *Vigna sinensis*, and others attacking plants related to the bean, each being confined rather closely to the species, or even to certain varieties within the species, to which they have adapted themselves.

At one time *Colletotrichum lagenarium* of the cucurbits was supposed to be identical with bean anthracnose, but later studies have shown that the two are distinct species.

There are numerous reports indicating varying degrees of susceptibility of the common bean varieties to anthracnose. Some have been

reported as resistant, while others have been listed as susceptible. In the light of recent investigations which have definitely established the occurrence of a number of physiological strains of the pathogene, many of these older reports are valueless. These physiological strains were recognized by Edgerton and Moreland (1916), and Barrus (1918) described two which he designated as *alpha* and *beta*. As a result of a comparative study of 300 varieties of beans and related species he recognized the following groups: (1) susceptible to both strains; (2) resistant to alpha, susceptible to beta; (3) susceptible to alpha, resistant to beta; (4) resistant to both strains. Group 1 included 80 per cent of the wax bush varieties, 50 per cent of the green-pod bush, 40 per cent of the wax pole and 30 per cent of the green-pod pole, which would show that, generally speaking, the wax bush beans are the most susceptible. Five varieties were placed in group 4, of which Wells' Red Kidney and White Imperial were considered the most important. The former was immune to *alpha* and resistant to *beta*, while the latter was resistant to *alpha* and practically immune to *beta*. Recently Burkholder (1923) has recognized a third biological strain originally isolated from the White Imperial, which he designates as *gamma*, and to which both Wells' Red Kidney and White Imperial are very susceptible. In a recent study Leach (1923) has recognized eight biological forms as a result of comparative inoculations with 15 cultures from different sources on 16 differential hosts. He concludes that these forms are relatively stable, that the difference in spore sizes of the biologic forms is of no practical significance, and that host resistance is constant. Muller (1926) has reported four different biological strains from Holland distinct from the American strains, while Budde (1928) by a study of 46 isolations from Germany, 2 from Sweden and 1 from Holland has recognized 9 biological strains, only one of which was similar to any of the American strains. This existence of numerous biological strains very greatly complicates the breeding of new varieties for resistance.

Some progress has been made in the production of anthracnose-resistant varieties by crossing and selection. Burkholder (1918) was able to produce an anthracnose-resistant White Marrow by crossing the common susceptible White Marrow with the resistant Wells' Red Kidney, and McRostie (1919, 1921) has accomplished similar results with the white pea bean, using Michigan Robust as the susceptible parent. They have shown that resistance to alpha, beta and gamma strains is dominant and governed by a single factor. Reddick (1928) has obtained some promising hybrids by crossing White Imperial, resistant to alpha and beta, and Robust, immune to mosaic, beta and gamma. To insure more definite success in obtaining resistant or immune varieties Ten Doornkaat-Koolman (1927) has inoculated seedlings of the F_2 generation with a mixture of all the biologic strains available.

Control.—A great many different control practices have been tried and recommended at various times, but most of them have proved either ineffective or only partially successful. The following may be mentioned: (1) seed disinfection; (2) sorting out the diseased seed by hand picking or by specific-gravity separation (flotation); (3) selecting seed from the field from anthracnose-free pods or maintaining a special seed plot for the production of the anthracnose-free seed; (4) regional selection or the procuring of seed from localities in which the anthracnose does not occur; (5) the avoidance of conditions favorable for infection or the dissemination of the spores and growth of the pathogene, such as close planting, especially in drills, low, moist sites, picking or cultivating when the plants are wet with rains or dew, planting at the time of the year most favorable to the disease; (6) reducing or removing the sources of infection by the removal of affected seedlings from the growing crop or by removing and destroying the diseased vines in the fall; (7) spraying plants in the field or applying a fungicide to the picked pods before shipping to market; (8) rotation of crops to avoid residual contaminations; and (9) the selection and breeding of disease-resistant varieties.

While nearly all of these measures have given a certain amount of protection and may under certain conditions be used with profit, first emphasis is placed on the production or use of anthracnose-free seed. Seed treatment with various disinfectants is ineffective and impractical because of the internal mycelium, which cannot be killed without injuring the seed, because soaking makes the seed unfit for machine planting unless dried, when many will slip their coats. Muncie (1917) obtained best results with a 30-minute immersion in a 35 per cent solution of bleaching powder, and by the formaldehyde sprinkle, but finally concluded that seed treatments with chemical solutions or with wet or dry heat were unsatisfactory. Spraying has yielded variable results in the hands of both growers and the experimenters, and many different fungicides have been tried. Some tests have been failures, while others have given a fair protection. Barrus (1921) concludes that

> Anthracnose and possibly blight may be kept in check by thoroughly spraying the plants with Bordeaux mixture, if the operation is begun soon after the plants have appeared above ground and continued at intervals of about 10 days until the pods are reaching marketable size.

With seed disinfection shown to be ineffective, spraying of doubtful value from the standpoint of expense and various other practices of minor importance, the production and use of disease-free seed and the use of resistant varieties must be the refuge of the bean grower.

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DIPLODIA DISEASE OF CORN

Diplodia zeæ (Schw.) Lev.

This is a disease which causes a seedling blight and a molding or rotting of the ears, but it also attacks the leaf sheaths and stalks of maturing plants. It has been called "mold," "moldy corn," "mildew," "rot" and "ear rot" and "dry rot," because of prominence of the ear effects; but since it also affects seedlings, the more general name of "Diplodia disease" is suggested.

History and Geographic Distribution.—Although the causal organism of dry rot was described by Schweinitz in his synopsis of Carolina fungi in 1834, it was not until 1906 that dry rot was recognized as a disease of corn (Heald). It first came to the attention of the writer during a trip to western Nebraska to investigate the death of horses that were being pastured in corn fields. It was found to be very common in that section and suspicion was at once directed to it as the possible cause of poisoning of the horses. While a feeding test that was at once carried out showed no injurious results after moldy corn had been used as the exclusive grain ration for 2 months, it was soon realized that this disease of corn was one of considerable importance in the corn-growing districts of the state. A study of the dry rot was made during the next 2 years and a preliminary report was published in 1908 (Heald, Wilcox and Pool). The prevalence of the disease in Illinois about the same time led to a study published by Burrill and Barrett a year later. The causal fungus was discussed by Reed in 1910 as a possible factor in the etiology of pellagra and this was followed by a study by the same investigator (1913) of the effect of *Diplodia zeæ* and some other fungi upon the phosphorus compounds of maize. In the earlier paper "data were presented showing that following the growth of the fungus a toxic substance was generated in the maize which was believed to be similar to or identical with the 'pellagrozein' of Lombroso," but the main result of the latter work was the establishment of the fact that the dry rot caused a marked deterioration in the food value of affected maize.

The disease was studied in South Africa in 1916 (Van der Bijl) and in 1919 Smith and Hedges presented evidence in favor of the systemic character of infections, but this has not been substantiated by later investigations. The unusual prevalence of the disease in Iowa in 1921 and 1922 induced a detailed study of the disease in that state (Durrell, 1920–1923) and later in that and adjacent states by several other workers. It had assumed importance in Australia by 1926 (Tryon). The trouble is known as far south as Florida and Texas and has been observed rarely in the Pacific Northwest, but seems to make its maximum development in the corn belt from Nebraska eastward. It is now recognized as one of the important ear rots of corn.

Symptoms and Effects.—The Diplodia disease of corn exhibits two rather distinct phases: (1) seedling blight; and (2) the infections on maturing plants, the most important feature being the dry rot of ears.

When corn from infected ears is used for seed, the stand may be materially reduced either by failure of grains to germinate or because the seedlings succumb to the attacks of the fungus before they emerge from the soil. In some tests infected seed has given only 50 per cent of the stand obtained with nearly disease-free seed (Raleigh, 1930). Evidence has also been presented to show that many seedlings from infected seed that do survive make weak plants that are low in their field performances with an increase in nubbins and barren stalks over normal, healthy plants, (Raleigh, 1930), which is contrary to the conclusions of Clayton (1927) that diseased seed did not increase barren, broken or leaning stalks. The competition between weak and normal plants in the same hill results in poorer production by the weak plants than when standing alone. According to Kiesselbach and Culbertson (1931), "there was no significant difference between the acre yields from diseased and healthy seed when the stand from the latter was adjusted to correspond with the former in number and distribution of plants." The stand reduction and the resultant effect on yield are the important consequences from the planting of infected seed.

In the early stages of dry rot of the ears or in case of light attacks, no external evidence of the presence of mold can be detected. The kernels in such light attacks may have a healthy appearance, but if they are removed or the ear is broken in two, a thin, whitish coating of mold may be found covering the tips of the kernels, or white flaky masses may be seen on the exposed surfaces at the end of the broken cob. In heavier infections or more advanced stages the fungus may completely cover the ears with a very evident mold, which may not be noticeable until the ear is husked, or in certain cases the mold may penetrate the husks, so as to be readily evident on the exterior, and the husks may be discolored and matted together by the infesting mold. Molded ears may be more or less discolored, being grayish or dirty looking or even brown or almost black. In the more advanced stages of the rot the following characters are prominent: (1) The ear is very light in weight (over 50 per cent loss), shriveled and frequently stands erect when healthy ears are pendent from

their weight; (2) the individual kernels are dull in color or have a dried brown appearance, are more brittle than normal ones and are more loosely attached to the cob; (3) minute black fruiting bodies may be found embedded in the flaky masses of mold between the kernels, in the sulci and on the surface of the corneous margins of the alveoli or on other parts of the cob, or even upon the husks or shanks of badly diseased ears. The presence of the black fruiting bodies is the most distinguishing mark of the disease, and should readily serve to separate the Diplodia or dry rot from

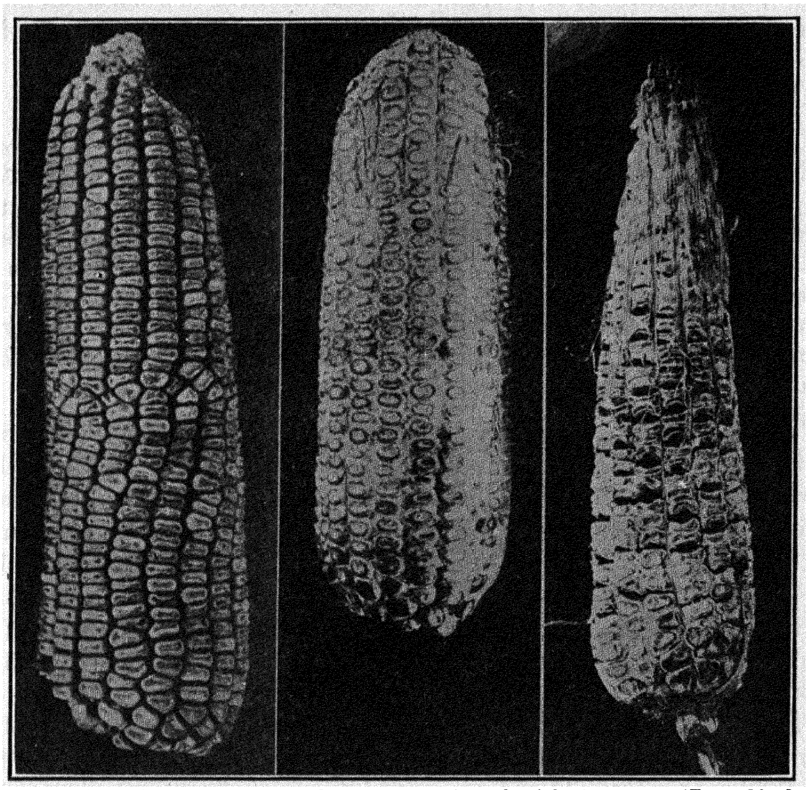

FIG. 203.—Normal ear of corn, and two ears affected with dry rot. (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

other ear rots of corn. It should be emphasized that ears may be very lightly infected and even show no evidence of the parasite until seed is tested on the germinator.

Early infections may cause small, shrunken ears of no value while less severe attacks produce ears of poor feeding quality. In severe attacks the embryos are destroyed and kernels have lost their viability, while in more moderate attacks the viability is lowered or impaired, so that the corn is of no value for seed purposes. The ear rot does not necessarily stop with the maturing of the corn, but may continue to advance even

after the corn is picked, especially if the ears are slightly soft or if they are subjected to moist conditions.

On the leaf sheaths the fungus produces reddish or purplish spots of varying size and shape, appearing after flowering of the corn plant. These lesions may extend down into the node of the stalk or up the leaf, killing and discoloring the midrib (Durrell, 1923).

This condition was first described by Burrill in 1889, under the name of the purple sheath spot of corn and was attributed to a bacterial pathogene, but Durrell has shown that the dry-rot organism is at least one of the causes of these discolorations.

This disease affects

. . . other parts of the corn plant besides the ear and sheaths. The shank of the ear is frequently affected and white wefts of mycelium may be seen on it. More common, however, is the appearance of large numbers of fruiting bodies at the shank and nodes. In some cases the breaking of the shank is due to the presence of Diplodia, though not always. On the stalk the symptoms are much like those on the shank. Within the sheath, or its base, and around the node may often be found a white growth of the mycelium. This may extend over the internode as well. Infection at the nodes, particularly the lower ones, is often manifested as a water-soaked discoloration (Durrell, 1923).

The effect of dry rot may be summarized as follows: (1) the reduction in yield, due to severely infected ears which are discarded at picking time, to weakened plants as a result of nodal infections and to poor stands which result from the use of Diplodia-infected seed; (2) the lowering in market quality due to the inclusion in the harvested crop of partly molded or slightly infected ears, which may suffer still further deterioration in storage; and (3) the lowering of quality for seed purposes because of the effect of the dry rot in reducing germination or causing seedling blight, thus causing thin stands (as much as 25 to 30 per cent reduction).

The importance of the disease in corn production may be realized from the study of Durrell, who reported that the losses in Iowa in 1921 and 1922 ranged from 3 to 15 per cent of the ears at harvest and caused an average damage to seed corn of 11 per cent. "A still further loss results from the nodal infection and weak plants grown from slightly infected seed."

Etiology.—Dry rot has been shown by pure culture inoculations to be due to *Diplodia zeæ* (Schw.) Lev., an imperfect fungus, which was formerly known only as a saprophyte on the old, dead stalks. Stevens records another species, *D. macrospora* Earle, from North Carolina, and this has recently been reported from Florida (Eddins, 1930) as causing a disease similar to *Diplodia zeæ*. In addition, ear rots may be caused by bacteria and several other species of fungi. It is not a systemic disease, but infection is local, the principal points of infection being the silks,

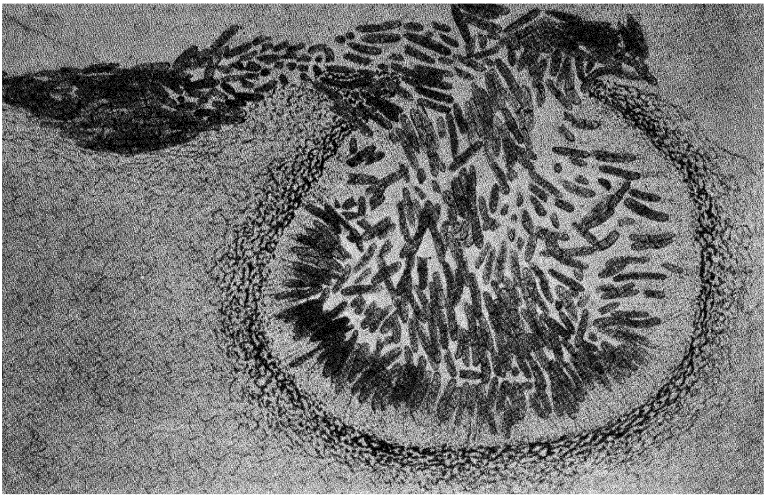

FIG. 204.—Pycnid of *Diplodia zeæ*, showing copious production of brown, one-septate spores. (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

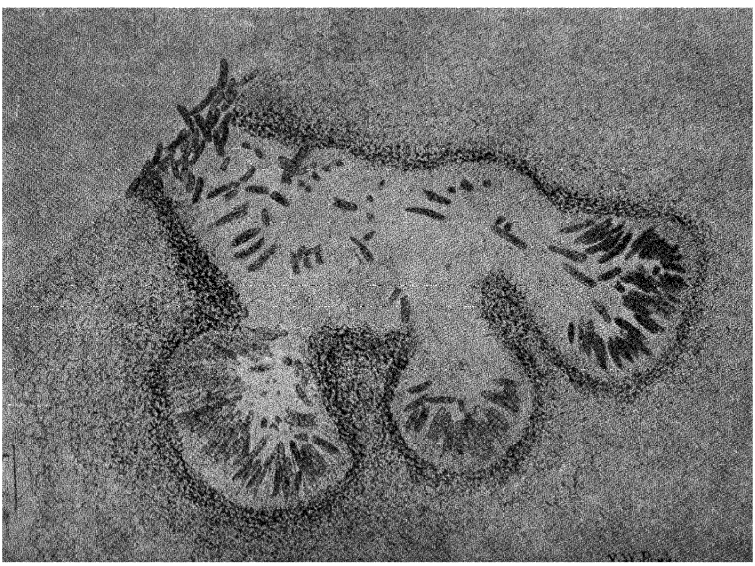

FIG. 205.—Pycnid of *Diplodia zeæ*, showing lobulated cavity. (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

tips of ears, ear shanks and nodes, the two latter being considered the chief points of attack. The fungus may spread through the tissues from its point of entrance, and thus ears may be pervaded by mycelium that has grown upward from the node or from the shank or downward from the silks or ear tip. Infections take place after flowering and the fungus continues to grow more rapidly in the languid tissues of the maturing plant parts, and later continues its saprophytic life on the old stalks during the late fall or during the next season.

The pathogene produces a single kind of fruiting body, *pycnidia*, which are the black bodies referred to in the descriptions of symptoms and effects. They are flattened spherical or pyriform, ostiolate, and produce large numbers of dark-brown, cylindrical to elliptical, obtuse, straight or usually curved, one- (rarely two-) septate pycnospores, 24 to 33 by 5 to 5.2μ. The pycnospores are extruded in amorphous masses or in tendrils which are apparently held together by a mucilaginous coating which readily dissolves in water causing their ready separation.

The pycnidia on the ears are most frequently seated in the dense masses of white mycelium from which they originate and are formed by the interwoven and fused masses of hyphæ which constitute the wall, from the inner surface of which numerous, simple, hyaline conidiophores are produced. These free pycnidia are generally larger and more irregular than those which are immersed in the tissues of stalks or other parts. They normally contain only a single locule, but in some cases this may be more or less lobulated with a common ostiole. The immersed pycnidia are vertically compressed or flattened in many cases, and may have thicker walls than those produced in the mycelial felt on the ears or in cultures.

The principal method of infection has been described as follows:

> Prior to the production of flowers by the corn plant the ligules of the leaf sheath clasp the stalk very tightly, preventing anything from slipping down inside the sheath. After flowering, however, the stalk has ceased elongating and the action of the wind on the leaves has by this time loosened the ligule and exposed the cavity of the sheath. At the same time masses of pollen fall and roll down the leaf into the sheath, together with such spores as they may carry along or which are blown in. In addition to the combination of the spores, and the stored food present in the pollen, a third factor of moisture enters. The sheaths of corn are frequently moist inside, even in dry weather holding condensed moisture in droplets, while in wet weather they stand full of water, and often remain that way days after a rain (Durrell, 1923).

The pycnospores germinate and at first feed on the food furnished by the moisture, pollen and exosmosed sugar, and when this is gone the hyphæ invade the tissues of the corn plant and cause the spots and blotches on the sheath which have previously been noted. These leaf-sheath invasions appear to be more frequent on the lower part of the

plants, due probably to earlier opening of the lower leaf sheaths and to more favorable moisture conditions. That infections can take place through the silks has been proved by artificial inoculations, and this is undoubtedly one of the ways by which infection occurs under natural conditions. Systemic infection from infected seed or systemic invasion through the roots has been suggested, but Durrell and others obtained no evidence of such behavior.

The principal method by which the pathogene is carried over the winter is undoubtedly in either the vegetative or fruiting condition on the old stalks or discarded ears. In the spring and summer old stalks bearing pycnids will show tendrils of spores following rain periods. In Illinois, "pieces of diseased stalks 1 and 2 years old have been found in July,

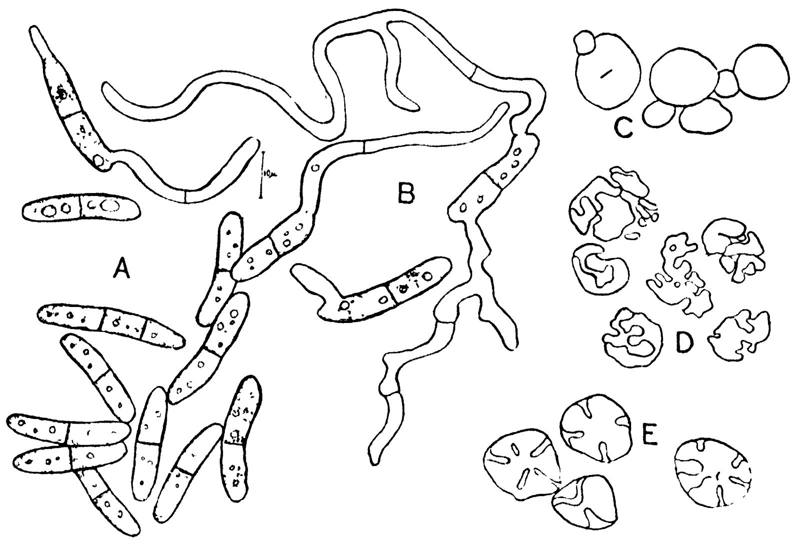

FIG. 206.—*A* and *B*, spores of Diplodia and their germination; *C*, normal starch grains in outline; *D*, partially digested starch grains from embryo of kernel affected by dry rot; *E*, partially digested starch grains from endosperm. (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

August and September almost covered with black tendrils of Diplodia spores capable of quick germination" (Burrill and Barrett, 1909), and stalks almost 3 years old were found to produce some few pycnospores capable of germinating. Mature spores germinate in from 5 to 8 hours or longer, in culture media, by sending out a germ tube from or near the distal end of each cell of the spore. The minimum temperature for growth is between 10 to 15°C., and the optimum between 28 and 30°C. It may be noted that favorable temperatures generally prevail in the corn belt at the season of the year when infections are probable.

Wind is believed to be the principal agent in the dissemination of the spores. By means of slide spore traps Burrill and Barrett demonstrated

that wind-borne pycnospores could be collected in considerable numbers at least 350 yards from an old field that had produced a diseased crop. While the spores may be carried to adjacent fields, a crop on contaminated ground will stand much better chances of infection. It has been shown that the fungus can grow in ordinary field soil containing organic matter, so it is possible that it can live in soil in a purely saprophytic manner, independent of old host remains.

The dry-rot fungus is an active feeder, and is reported to produce numerous enzymes by which it digests the constituents of the host tissue which it permeates. It may be noted that it has the power to corrode and digest starch grains, and the

> . . . utilization of cellulose by the fungus is of particular interest, as the penetration and growth in the stalk and weakening of the nodes are thus more readily understood. The survival of the organism in old stubble and trash, even though plowed under for several seasons, appears probable (Durrell, 1923).

The pathogene can penetrate the cloth of rag-doll germinators and infect kernels of adjacent ears. It requires air for its growth, hence cannot be a factor in the spoiling or deterioration of ensilage which is cured under anaerobic conditions.

Conditions Favoring Dry Rot.—Since dry rot appears in unusual amount in certain seasons only, its development must bear some relation to certain environmental factors. The studies of Durrell have shown that the moisture relation is the most important factor. The development of the disease in the field appears to be greatly favored by heavy rains in August and September, or during the period when the corn is approaching maturity. While abundant moisture earlier in the season may cause a few infections, later rains have the greatest effect.

> Certain other conditions must also accompany high precipitation in order to produce an epidemic of dry rot. These other conditions exist annually toward the end of the growing season and are as follows: First, there is a maximum of stored food in the corn plant; second, the rapid growth of tissues has ceased and the leaf sheaths have become loosened; third, the loose leaf sheaths afford lodgment for spores of *Diplodia zeæ*. The presence of water between the leaf sheaths and the stalk at this time initiates infection. In seasons when a plentiful water supply is available during this period (August), dry rot is prevalent (Durrell, 1923).

The later moisture relations are also of importance. In regions in which Diplodia is prevalent, it has repeatedly been observed that many ears may be infected without showing any external evidence of the disease. Such infected ears are, of course, harvested along with the disease-free ears. If infected corn that is still high in moisture content is stored so that it cannot dry out, or in open cribs exposed to rain, the dry rot will advance rapidly, especially if warm weather prevails. If the corn con-

tains sufficient moisture there will be a slow advance of the dry rot during the cooler periods, since growth will not be prevented until temperatures of 10 to 15°C. have been reached.

It has recently been emphasized that "Diplodia-infected seed may result in a poor stand, especially when the soil is cool and moderately wet." In a comparison of tests at two temperatures, 20 to 24°C. and 15 to 19°C., an important effect of temperatures has been noted (Raleigh, 1930).

Although just as many kernels germinated at the low temperature, the stand or number of plants emerged was greater at the higher temperature. On examination, it was found that many of the infected plants were killed before they reached the surface. More plants showed lesions, and the lesions were more extensive at the higher than at the lower temperature.

These results are in accord with field experiments at Ames and with those of Holbert, Burlison, *et al.* (1924), in that better stands were obtained from Diplodia-infected seed in warm than in cold soils. They are not in accordance with the conclusions of Durrell (1923) and Clayton (1927) in their statements that seedling blight from seed infected with *Diplodia zeæ* is not important at the relatively cool temperatures prevalent in the spring.

Control.—In considering the prevention of this disease or reducing its damage to the least possible amount, the following features should be kept in mind: (1) The fungus on the old stalks is the principal source of the new infections; (2) the spores can be carried by the wind from a contaminated field to healthy plants in an adjacent field; (3) stalks 2 or 3 years old which harbor the pathogene are still capable of producing viable spores; (4) seed corn may be infected with dry rot without external evidences of the disease; (5) such infected seed may give poor stands owing to failure to germinate or the plants which do develop may show a decreased height, slenderer stalks and a poorly colored foliage.

In the light of the above, the control measures which are suggested are sanitation, crop rotation, care in curing and storing the harvested crop and extra care in the selection, storing and testing of seed corn and seed disinfection.

The sanitary practices which might be adopted consist of the collection and destruction of the rot-infected ears at time of husking, the low cutting and hauling away from the field of the stalks or their destruction by burning or the avoidance of immediate return of manure that may contain infected stalks to ground that is to be planted to corn. Considering the persistence of the pathogene on the old stalks, a 4- or 5-year rotation is more desirable than a short one. It is possible that, with such a long rotation and cultural practices designed to hasten the decay of the stalks, the sanitary practices might be ignored, but in case of a short rotation the amount of inoculum should be reduced as much as possible. "Care should also be taken not to plant corn by the side of an old infected

field, especially if the latter is upon the side from which come the prevailing summer winds" (Burrill and Barrett, 1909).

The seed corn should be selected as soon as the ears are well filled and dented without regard to the time of frost. Seed ears should only be selected from green standing stalks with sound shanks. After the seed has been gathered it should be cured in a dry, well-ventilated place, not exposed to the weather (Durrell, 1923).

Seed disinfection will not affect the amount of ear rot appearing in a field, since it has been shown that the use of infected seed does not increase the amount of ear rot. Seed disinfection of infected seed has been shown to increase the stand and lessen the amount of seedling blight (Reddy, Holbert and Irwin, 1926; Reddy and Holbert, 1928; Melhus *et al.*, 1928; Kiesselbach, 1931). Thorough dusting of Diplodia-infected seed with Merko, Bayer dust or Semesan Jr. has given increases in yield from 22 to 30 per cent (Raleigh, 1930).

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OTHER IMPORTANT DISEASES DUE TO IMPERFECT FUNGI

DUE TO SPECIES OF MONILIALES

Blackheart or rot of pineapple (*Thielaviopsis paradoxa* (d. Seyn.) V. Höhn.).—Causes a destructive rot beginning at the stem end. PATTERSON, FLORA W., CHARLES, VERA K. AND VEIHMEYER, F. J.: II. Pineapple rot caused by *Thielaviopsis paradoxa*. *U. S. Dept. Agr., Bur. Pl. Ind. Bul.* **171**: 15–35. 1910. HOWARD, A.: On Some diseases of sugar cane in the West Indies. *Ann. Bot.* **17**: 373–401. 1903. ROLDAU, E. F.: The soft rot of pineapple in the Philippines and other countries. *Philippine Agr.* **13**: 397–405. 1925. DADE, H. A.: *Ceratostomella paradoxa*, the perfect stage of *Thielaviopsis paradoxa* (DeSeynes) Von Höhnel. *Trans. Brit. Myc. Soc.* **13**: 184–194. 1928. See also Pineapple disease of sugar cane, p. 658.

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Texas root rot of cotton (*Phymatotrichum omnivorum* (Shear) Duggar).—This disease is characterized by a sudden wilting of affected plants, very commonly without previous dwarfing or without any warning symptoms of chlorosis. The foliage droops, turns brown and may remain hanging for some days and later drop off, leaving the bare, dead stalk. If the root of such an affected plant is carefully removed from the soil, the causal fungus will be evident on its surface as numerous yellowish or tawny strands or threads. The cortex of the killed root is soft and readily peels off from the central woody cylinder. Under certain conditions, especially of abundant moisture, wart-like bodies, or pseudosclerotia, may be present on the surface of affected roots. The disease generally appears in spots in the field and these may be circular or of varying form. Spots affected one year may not necessarily produce a diseased crop the next season. The root-rot fungus was considered as a sterile form until the conidial stage was described by Duggar (1916). This conidial stage was collected by the writer at Falfurrias, Tex., in 1910, but was not at that time connected with the typical Texas

root rot. The conidial stage develops on the ground near the margin of a zone of dying plants in the form of cushion-like, creamy or yellowish masses. This trouble on its various hosts is serious in Texas and occurs to a lesser extent in Oklahoma, New Mexico, Arizona, Mexico and Southern California but is absent from the regions east of the Mississippi. According to Taubenhaus (1923), it is "capable of attacking 31 different economic field crops, 58 different truck crops, 18 different kinds of fruits and berries, 35 different kinds of forest trees and shrubbery, 7 different kinds of outdoor herbaceous ornamentals and 20 different kinds of weeds." The loss from root rot in Texas was estimated in 1920 at 630,000 bales, or 15 per cent of the entire crop. The sources of loss are: (*a*) plants killed without reaching maturity; (*b*) reduced yield and inferior quality from plants which are killed when the bolls are partly developed; and (*c*) inferior quality of the lint from affected plants which remain alive during the entire growing season. The fungus is believed to winter over only on the roots of a living host. DUGGAR, B. M.: The Texas root-rot fungus and its conidial stage. *Ann. Mo. Bot. Gard.* **3**: 11–23. 1916. TAUBENHAUS, J. J. AND KILLOUGH, D. T.: Texas root rot of cotton and methods of its control. *Tex. Agr. Exp. Sta. Bul.* **307**: 1–98. 1923. KING, C. J.: Habits of the cotton root-rot fungus. *Jour. Agr. Res.* **26**: 405–418. 1923. PELTIER, G. L., KING, C. J. AND SAMSON, R. W.: Ozonium root rot. *U. S. Dept. Agr. Bul.* **1417**: 1–27. 1926. TAUBENHAUS, J. J., DANA, B. F. AND WOLFF, S. E.: Plants susceptible or resistant to cotton root rot and their relation to control. *Tex. Agr. Exp. Sta. Bul.* **393**: 1–30. 1929. MCNAMARA, H. C., HOLTEN, D. R. AND PORTER, D. D.: Cycles of growth in cotton root rot at Greenville, Tex. *U. S. Dept. Agr. Circ.* **173**: 1–17. 1931. (A basidial stage of the Texas root-rot fungus has been described as *Hydnum omnivorum*. See p. 854.)

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Tomato wilt (*Fusarium lycopersici* Sacc.).—Wilt of tomatoes is a very serious disease, especially in the Middle Atlantic, Gulf and lower Mississippi Valley states, but is also prevalent in other sections from the Atlantic to the Pacific. It is characterized by a yellowing, an upward and inward rolling of the leaves and wilting

followed by death. Cross and longitudinal sections, especially at the base of the stem of infected plants, show a brown discoloration of the vascular bundles. The pathogene may also advance into leaves, fruit pedicels, fruits and even into the seeds. Much progress has been made in the development of wilt-resistant strains. EDGERTON, C. W. AND MORELAND, C. C.: Tomato wilt. *La. Agr. Exp. Sta. Bul.* **174**: 1–54. 1920. ELLIOTT, J. A. AND CRAWFORD, R. F.: The spread of tomato wilt by infected seed. *Phytopath.* **12**: 428–434. 1922. PRITCHARD, F. J.: Development of wilt resistant tomatoes. *U. S. Dept. Agr. Bul.* **1015**: 1–18. 1922. CLAYTON, E. E.: The relation of temperature to the development of Fusarium wilt of the tomato. *Amer. Jour. Bot.* **10**: 71–88. 1923. SCOTT, I. T.: The influence of hydrogen-ion concentration on the growth of *Fusarium lycopersici* and on tomato wilt. *Mo. Agr. Exp. Sta. Res. Bul.* **64**: 1–32. 1924. WHITE, R. P.: Studies on tomato wilt caused by *Fusarium lycopersici* Sacc. *Jour. Agr. Res.* **34**: 197–239. 1927. HAYMAKER, H. H.: Relation of toxic excretory products from two strains of *Fusarium lycopersici* to tomato wilts. *Jour. Agr. Res.* **36**: 697–719. 1928. MCWHORTER, F. P. AND PARKER, M. F.: A comparison of wilt-resistant tomatoes in Virginia. *Va. Truck Exp. Sta. Bul.* **69**: 789–797. 1929.

Fusarium wilt or blight of the potato (*Fusarium oxysporum* Schlecht).—This widespread soil fungus can attack any part of the potato plant and produce a wilting and death of the plant with or without rotting of roots, stems or tubers. Neither the vascular discoloration in the stem nor the stem-end browning of tubers is a diagnostic character of the disease, since the symptom may be absent in wilt or may develop as a result of drought and high temperatures. Infection may take place through roots or seed pieces from the organism already in the soil or from infected seed, but the former method is the more frequent. Control of the disease cannot be accomplished by cutting off the discolored stem ends of seed tubers, but such discolored tubers are objectionable for seed purposes, as they are likely to produce weak plants. MACMILLAN, H. G.: Fusarium blight of potatoes under irrigation. *Jour. Agr. Res.* **16**: 279–303. 1919. BISBY, G. R.: Studies on Fusarium diseases of potatoes and truck crops in Minnesota. *Minn. Agr. Exp. Sta. Bul.* **181**: 1–44. 1919. EDSON, H. A.: Vascular discoloration of Irish potato tubers. *Jour. Agr. Res.* **20**: 277–294. 1920. MCKAY, M. B.: Transmission of some wilt diseases in seed potatoes. *Jour. Agr. Res.* **21**: 821–848. 1921. GOSS, R. W.: Relation of environment and other factors to potato wilt caused by *Fusarium oxysporum*. *Neb. Agr. Exp. Sta. Res. Bul.* **23**: 1–84. 1923. FAHMY, T.: The production by *Fusarium solani* of a toxic excretory substance capable of causing wilting in plants. *Phytopath.* **13**: 543–550. 1923. MCKAY, M. B.: Potato wilt and its control. *Ore. Agr. Exp. Sta. Bul.* **221**: 1–23. 1926.

Wilt of cotton (*Fusarium vasinfectum* Atk.).—This disease of cotton frequently causes dwarfing, but discoloration of leaves, wilting and death of plants are responsible for heavy losses in the southeastern United States. The trouble generally appears in spots in a field and these may enlarge from year to year. Young plants may die or they may succumb at various stages of their growth. The disease has been called "Black root," since the roots of affected plants turn black. A brown or a black discoloration of the xylem in stems or other affected parts is a strong evidence of the disease. Wilt of cotton, watermelons and cowpeas was attributed to an ascigerous fungus, *Neocosmospora vasinfecta* (Atk.) Smith, but more recent investigations have shown that the ascigerous fruits belonged to a saprophytic intruder (Higgins, 1911). Crop rotation and the use of wilt-resistant varieties are the accepted methods of control. Federal and state departments and private individuals have bred resistant varieties. The first production of the

Bureau of Plant Industry was Dillon, but this has been largely replaced by Dixie, a more desirable variety. SMITH, ERWIN F.: Wilt disease of cotton, watermelon and cowpea. *U. S. Dept. Agr., Div. Veg. Phys. & Path. Bul.* **17**: 1–53. 1899. HIGGINS, B. B.: Is *Neocosmospora vasinfecta* (Atk.) Smith the perithecial stage of the Fusarium which causes cowpea wilt? *N. C. Agr. Exp. Sta. Ann. Rept.* **32**: 100–116. 1911. LEWIS, A. C. AND MCLENDON, E. A.: Cotton variety tests for boll weevil and wilt conditions in Georgia. *Ga. State Bd. Ent. Bul.* **46**: 1–36. 1917. GILBERT, W. W.: Cotton diseases and their control. *U. S. Dept. Agr., Farmers' Bul.* **1187**: 1–32. 1921. ELLIOTT, J. A.: Cotton wilt, a seed-borne disease. *Jour. Agr. Res.* **23**: 387–393. 1923. NEAL, D. C.: Cotton wilt: a pathological and physiological investigation. *Ann. Mo. Bot. Gard.* **14**: 359–424. 1927. YOUNG, V. H.: Cotton-wilt studies I. *Ark. Agr. Exp. Sta. Bul.* **226**: 1–50. 1928. ——, WARE, J. O. AND JANSSEN, G.: Cotton wilt studies II. *Ark. Agr. Exp. Sta. Bul.* **234**: 1–32. 1929. DASTUR, J. F.: Cotton wilt. *Mem. Dept. Agr. India Bot. Ser.* **17**: 29–73. 1929. WALKER, M. N.: Potash in relation to cotton wilt. *Fla. Agr. Exp. Sta. Bul.* **213**: 1–10. 1930.

DUE TO SPECIES OF MELANCONIALES

Anthracnose of grape (*Glœosporium ampelophagum* Sacc.).—The cause of stem lesions and fruit infections giving rise to the popular name of "bird's-eye disease." SHEAR, C. L.: Grape anthracnose in America. *Int. Cong. Vit. Rept.* **1915**: 111–117. 1916. CASTELLA, F. DE AND BRITTLEBANK, C. C.: Anthracnose or black spot of the vine. *Jour. Dept. Agr. Victoria* **15**: 404–421. 1917. Reported ascigerous stage *Manginia ampelina* (V. & P.). SHEAR, C. L.: The life history of *Sphaceloma ampelinum* (De B.). *Phytopath.* **19**: 673–679. The ascigerous stage is described as *Elsinœ ampelina*, a species very similar to the Rubus form, *Elsinœ veneta* (Plectodiscella).

Citrus scab (*Glœosporium fawcettii* (Jenkins).—This is an important citrus disease of the Gulf states and many foreign countries, which produces its characteristic lesions on leaves and fruits, and to a lesser extent on young succulent twigs. WINSTON, J. R.: Citrus scab: Its cause and control. *U. S. Dept. Agr. Bul.* **1118**: 1–38. 1923. PELTIER, GEORGE L. AND FREDERICH, W. J.: Relation of environmental factors to citrus scab caused by *Cladosporium citri* Massee. *Jour. Agr. Res.* **28**: 241–254. 1924. JENKINS, ANNA E.: The citrus-scab fungus. *Phytopath.* **15**: 99–104. 1925. Changed from *Cladosporium citri* Fawcett (not Massee) to the form genus Sphaceloma. CUNNINGHAM, H. S.: Histology of the lesions produced by *Sphaceloma fawcetii* Jenkins on leaves of citrus. *Phytopath.* **18**: 539–545. 1928. JENKINS, ANNA E.: Insects as possible carriers of the citrus-scab fungus. *Phytopath.* **20**: 345–351. 1930.

Superficial bark canker of apple (*Myxosporium corticolum* Edg.).—Common as the cause of cankers on apples and pears in northeastern states. EDGERTON, C. W.: Two little-known Myxosporiums. *Ann. Mycol.* **6**: 47–52. 1908. GILCHRIST, G. G.: Bark-canker disease of apple trees caused by *Myxosporium corticolum* Edg. *Trans. Brit. Myc. Soc.* **8**: 230–243. 1923.

Anthracnose of cucurbits (*Colletotrichum lagenarium* (Pass.) Ell. & Hals.).—This disease is characterized by discolored or sunken spots on leaves, stems and fruits of cucumbers, muskmelons and watermelons, besides several non-economic hosts. GARDNER, M. W.: Anthracnose of cucurbits. *U. S. Dept. Agr. Bul.* **727**: 1–68. 1918. STEVENS, F. L. The ascigerous stage of *Colletotrichum lagenarium* induced by ultra-violet radiation. *Mycologia.* **23**: 134–139. 1931. Reported as *Glomerella lagenarium*.

Wither tip of citrus plants (*Colletotrichum glœosporioides* Penz.).—This trouble affects leaves, twigs and fruits. In all citrus fruits except limes it is connected with a

dying back of twigs and branches. It has been reported to cause much of the tear staining or tear streaking of various varieties, but this relation has been disputed and tear stain, at least under Florida conditions, is attributed to the work of rust mites. It is also associated with the anthracnose spotting and storage rot of fruit. FAWCETT, H. S.: Citrus diseases of Florida and Cuba compared with those of California. *Cal. Agr. Exp. Sta. Bul.* **262**: 153–210. BURGER, O. F.: Variations in *Colletotrichum glœosporioides*. *Jour. Agr. Res.* **20**: 723–736. 1921. WINSTON, J. R.: Tear stain of citrus fruits. *U. S. Dept. Agr. Bul.* **924**: 1–12. 1921. FAWCETT, H. S. AND LEE, H. A.: *In* Citrus Disease and Their Control, pp. 287–293; 396–398; 454–461. McGraw-Hill Book Company, Inc., New York. 1926.

Anthracnose or ring spot of lettuce (*Marssonina panattoniana* (Berl.) Magn.).—This disease is characterized by brownish sunken spots on midribs and petioles of the leaves and by dead spots in the leaf blade, which soon fall away, leaving perforations. The disease appears in the field and under glass and is more severe in a fall-seeded crop than in spring seedlings. The pathogene has been found on overwintering rosettes of wild lettuce (*Lactuca scariola*). BRANDES, E. W.: *Jour. Agr. Res.* **13**: 261–280. 1918. SALMON, E. S. AND WORMALD, H.: The "ring spot" and rust disease of lettuce. *Jour. Min. Agr. Gt. Brit.* **30**: 147–151. 1923. PAPE, H.: Der Pilze, *Marssonia panattoniana* Berl. als Schädling des Samensalates. *Gartenbauwissensch.* **1**: 524–527. 1929.

Blight of stone fruits (*Coryneum beijerinckii* Oud.).—This trouble has sometimes been called California blight because it was first studied in this country in California. It is probably identical with the pustular spot of the eastern United States and has long been known in Europe and other foreign countries. It causes a leaf spot and shot-holing twig spots on one-year-old shoots, with frequent invasion of adjacent buds, cankers on older branches and spotting or rotting of fruit. The final result may be more or less defoliation, dieback of shoots and dropping or disfiguring of the fruit. It affects peaches, apricots, plums and cherries. Fruit lesions on cherries differ from those on peaches and apricots by being fewer (one or two) and causing a drying of the tissue to the pit or a spreading infection that involves the entire fruit or a large portion of it. SMITH, R. E.: California peach blight. *Cal. Agr. Exp. Sta. Bul.* **191**: 73–98. 1907. CORDLEY, A. B. AND CATE, C. C.: Spraying for peach fruit spot. *Ore. Agr. Exp. Sta. Bul.* **106**: 4–15. 1909. PARKER, CHARLES: Coryneum blight of stone fruits. *Howard Rev.* **2**: 1–40. 1925. SAMUEL, G.: On the shot-hole disease caused by *Clasterosporium carpophilum* and on the shot-hole effect. *Ann. Bot.* **41**: 375–404. 1928.

Leaf spot of cherries and plums (*Cylindrosporium spp.*).—These imperfect fungi are stages in the life cycle of *Coccomyces sps.* (See special treatment of Cherry Leaf Spot, p. 551.)

DUE TO SPECIES OF SPHÆROPSIDALES

Apple blotch (*Phyllosticta solitaria* E. & E.).—This important apple disease of the Ozarks and the central Mississippi Valley is characterized by the production of lesions on leaves, fruits and spurs, twigs or branches. The leaf spots are small, irregular or angular, light brown or yellowish, 1 to 2 millimeters in diameter and each produces one to several pycnidia. The fruit lesions appear as dark or nearly black blotches marked by irregular, jagged or fringed margins. Minute black pycnidia appear in the center of the blotch. Minute discolored spots each bearing several pycnidia appear on the 1-year-old wood. On the older branches the bark of the cankers is roughened and cracked. SCOTT, W. W., AND ROHER, J. B.: Apple blotch, a serious disease of southern orchards. *U. S. Dept. Agr.*,

Bur. Pl. Ind. Bul. **144**: 1–28. 1909. LEWIS, D. E.: The control of apple blotch. *Kan. Agr. Exp. Sta. Bul.* **196**: 520–574. 1913. ROBERTS, J. W.: Apple blotch and its control. *U. S. Dept. Agr. Bul.* **534**: 1–11. 1917. SELBY, A. D.: Apple blotch, a serious fruit disease. *Ohio Agr. Exp. Sta. Bul.* **333**: 487–505. 1919. GARDNER, M. W., GREENE, L. AND BAKER, C. E.: Apple blotch. *Ind. Agr. Exp. Sta. Bul.* **267**: 1–32. 1923. GUBA, E. F.: Phyllosticta leaf-spot, fruit-blotch and canker of the apple; its etiology and control. *Phytopath.* **14**: 234–237. 1924. Also *Ill. Agr. Exp. Sta. Bul.* **256**: 481–551. 1925. ——: Pathologic histology of apple blotch. *Phytopath.* **14**: 558–568. 1924. ROBERTS, J. W. AND PIERCE, L.: Apple blotch. *U. S. Dept. Agr., Farmers' Bul.* **1479**: 1–11. 1926. McCLINTOCK, J. A.: The longevity of *Phyllosticta solitaria* (E. & E.) on apple seedlings held in cold storage. *Phytopath.* **20**: 841–848. 1930.

Fruit spot of apple (*Phoma pomi* Pass.).—This disease is characterized by the formation of minute spots, generally centering at the fruit lenticels. When young, the lesions are of the same color as the skin of the fruit but darker, while with age they become dark red, brown or black and somewhat sunken. The lesions are relatively superficial but with age cause a discoloration of the tissue beneath the skin, and minute spore fruits appear in the center of the spot or are radially arranged. The causal fungus first produces a Cylindrosporium stage, which is followed by the Phoma stage. More recently a perfect stage has been described (Walton and Orton, 1926) and referred to *Mycosphærella pomi* Passer. The fruit spot is a disease that is rather general throughout the northeastern United States. BROOKS, CHARLES: The fruit spot of apples. *N. H. Agr. Exp. Sta.* Rept. **1920**: 332–365. 1908. —— AND BLACK, C. A.: Apple fruit spot and quince blotch. *Phytopath.* **2**: 63–72. 1912. THOMAS, R. C.: A new fruit spot of the apple, Brooks' spot. *Ohio Agr. Exp. Sta. Mo. Bul.* **8**: 91–96. 1923. WALTON, R. C. AND ORTON, C. R.: The perfect stage of *Cylindrosporium pomi*. *Science* **63**: 236. 1926. MARTIN, H. W.: The Phoma fruit spot of the apple. *Proc. New Jersey State Hort. Soc.* **1929**: 57–66. 1930.

Blackleg of crucifers (*Phoma lingam* (Tode) Desm.).—Lesions appear on the stems near the ground and these soon become sunken and dark, hence the name blackleg. Lesions may be formed on petioles and leaves, and these generally bear numerous minute black pycnidia. Seedlings may collapse while still in the seed bed, or the disease may not be apparent until the plants have become established in the field. The pathogene persists in the soil and may be carried by the seed. HENDERSON, M. P.: The blackleg disease of cabbage caused by *Phoma lingam* (Tode) Desm. *Phytopath.* **8**: 379–431. 1918. WALKER, J. C.: Seed treatment and rainfall in relation to the control of blackleg. *U. S. Dept. Agr. Bul.* **1029**: 1–27. 1922. ——: The hot-water treatment of cabbage seed. *Phytopath.* **13**: 251–253. 1923. REX, E. G.: Experiments on the control of blackleg disease of cabbage. *Pa. Agr. Exp. Sta. Bul.* **199**: 1–23. 1925. CLAYTON, E. E.: Blackleg disease of brussels sprouts, cabbage and cauliflower. *N. Y. State Agr. Exp. Sta. Bul.* **550**: 1–27. 1927. ——: Seed treatment for blackleg disease of crucifers. *N. Y. (Geneva) Agr. Exp. Sta. Tech. Bul.* **137**: 7–58. 1928.

Tomato Fruit Rot (*Phoma destructiva* Plow.).—This disease is of most concern because of its effect on the fruits, but leaf spots also accompany the trouble. Lesions appear on either green or ripe fruit, and are evident as brown or almost blackish depressed spots 1 to 3 centimeters in diameter, upon which dark pycnidia may be formed. On fruit in storage or in transit to market, the causal fungus may form a superficial growth of dirty-gray, fluffy mycelium and the fruits may be ruined by the spread of the pathogene. This fruit rot is most prevalent in Cuba and the southern United States, and frequently causes heavy losses on shipments to the northern states. JAMIESON, C. O.: *Phoma destructiva*, the cause of a fruit rot

of the tomato. *Jour. Agr. Res.* **4**: 1–20. 1915. LINK, G. K. K. AND MEIER, F. C.: Phoma rot of tomatoes. *U. S. Dept. Agr. Circ.* **219**: 1–5. 1922.

Eggplant Blight (*Phomopsis vexans* (Sacc. & Syd.) Hart.).—Three types of injury are caused by this disease: (*a*) damping-off or seedling stem blight of young seedlings due to attacks of the fungus for an inch or more above the soil level; (*b*) brown leaf spots, 2 millimeters to 2 to 3 centimeters in diameter, of round, oval, oblong or irregular shape; (*c*) fruit lesions appearing at first as circular, depressed, discolored areas of rotted tissue which may advance rapidly until the entire fruit is rotted. Pycnidia may appear on the killed tissues and are especially abundant in the fruit lesions, appearing first at the center and then over the entire surface of the fruit, which later becomes a more or less shriveled black mummy. Two types of spores are produced: (*a*) pycnospores of the Phoma type, 5 to 8 by 2 to 2.8μ and two-guttulate; and (*b*) filiform, curved stylospores 13 by 28μ. HARTER, L. L.: Fruit rot, leaf spot, and stem blight of the eggplant caused by *Phomopsis vexans*. *Jour. Agr. Res.* **2**: 331–338. 1914. EDGERTON, C. W. AND MORELAND, C. C.: Eggplant blight. *La. Agr. Exp. Sta. Bul.* **178**: 1–44. 1921. NOLLA, J. A. B.: The eggplant blight and fruit rot in Porto Rico. *Jour. Dept. Agr. Porto Rico* **13**: 36–57. 1929.

Melanose and stem-end rot of citrus fruits (*Phomopsis citri* Faw.).—Both of these troubles are due to the same fungus, melanose resulting at any time when conditions are favorable and there is a flush of growth, while stem-end rot occurs only when the fruit is approaching maturity or has reached maturity. Melanose has the same general appearance on leaves, stems or fruits and results in the formation of "small hard, raised, reddish-brown spots or specks, scattered over the surface of leaves or fruits. In general, these spots are round with a smooth, glazed surface. In slight attacks the spots may be so widely scattered as to escape notice, while in severe cases the surfaces may be thickly studded with small spots or specks" (Stevens, 1918). The stem-end rot leads to injury to the fruit in two ways: (*a*) by causing dropping and rot either before or after maturity on the tree; and (*b*) by causing a softening and rot in transit to market or while being held for sale. "Next to the blue and green molds, this decay is the most common and troublesome rot in Florida during certain seasons, when conditions are right for its development" (Fawcett, 1915). FAWCETT, H. S.: Stem-end rot of citrus fruits. *Fla. Agr. Exp. Sta. Bul.* **107**: 1–23. 1911. FLOYD, B. F. AND STEVENS, H. E.: Melanose and stem-end rot. *Fla. Agr. Exp. Sta. Bul.* **111**: 1–16. 1912. FAWCETT, H. S.: Citrus diseases of Florida and Cuba compared with those of California. *Cal. Agr. Exp. Sta. Bul.* **262**: 151–211. 1915. STEVENS, H. E.: Melanose II. *Fla. Agr. Exp. Sta. Bul.* **145**: 102–116. 1918. WINSTON, J. R. AND BOWMAN, J. J.: Commercial control of melanose. *U. S. Dept. Agr. Circ.* **259**: 1–8. 1923. WINSTON, J. R., FULTON, HARRY, R. AND BOWMAN, J. J.: Commercial control of citrus stem-end rot. *U. S. Dept. Agr. Circ.* 293: 1–10. 1923. BURGER, O. F., DEBUSK, E. F. AND BRIGGS, W. R.: Preliminary report on controlling melanose and preparing Bordeaux-oil. *Fla. Agr. Exp. Sta. Bul.* **167**: 123–140. 1923. WOLF, F. A.: The perfect stage of the fungus which causes melanose of citrus. *Jour. Agr. Res.* **33**: 621–625. 1926. The ascogenous stage is *Diaporthe citri*. WINSTON, J. R., BOWMAN, J. J. AND BACH, W. J.: Citrus melanose and its control. *U. S. Dept. Agr. Bul.* **1474**: 1–62. 1927. BACH, W. J. AND WOLF, F. A.: The isolation of the fungus that causes citrus melanose and the pathological anatomy of the host. *Jour. Agr. Res.* **37**: 243–252. 1928.

End rot of cranberries (*Fusicoccum putrefaciens* Shear).—The rot caused by this organism generally begins at either the stem end or the blossom end, hence was named the end rot. In the inception of the trouble there is a softening of the

tissues accompanied by discolorations, and affected fruits are finally transformed into dark, dry, shriveled mummies. It is a serious storage rot of the cranberry. SHEAR, C. L.: End rot of cranberries. *Jour. Agr. Res.* **11**: 35–42. 1917. STEVENS, N. E.: Temperatures of the cranberry regions of the United States in relation to the growth of certain fungi. *Jour. Agr. Res.* **11**: 521–529. 1917. —— AND MORSE, F. W.: The effect of the end-rot fungus on cranberries. *Amer. Jour. Bot.* **6**: 235–241. 1919. SHEAR, C. L. AND BAIN, H. F.: Life history and pathological aspects of *Godronia cassandræ* Peck. (*Fusicoccum putrefaciens* Shear) on cranberry. *Phytopath.* **19**: 1017–1024. 1929.

Canker of poplar and willow (*Cytospora chrysosperma* (Pers.) Fr.).—Typical cankers are formed on twigs, limbs or trunks which may be girdled. Pycnidia are formed in abundance, and there is a copious protrusion of reddish-yellow spore horns following rains or during humid periods. The pathogene affects both poplars and willows. LONG, W. H.: An undescribed canker of poplars and willows caused by *Cytospora chrysosperma*. *Jour. Agr. Res.*, **13**: 331–345. 1918. POVAH, A. H. W.: An attack of poplar canker following fire injury. *Phytopath.* **11**: 157–165. 1921.

Citrus knot (*Sphæropsis tumefaciens* Hedges).—In this trouble of limes and oranges, knots varying from ⅜ to 2 or 3 inches in diameter may be formed on twigs or branches. The galls are generally round, but in some cases an eruption may be formed extending parallel to the axis of a branch for several inches. The galls are at first smooth but with age become rough and fissured. Groups of branches may be formed from the knots on lime trees, giving rise to typical witches' brooms, the shoots of which may also develop knots. Pycnids of the fungus develop on the knots or on the dead bark of adjacent parts. The disease was first reported from Jamaica but has since been studied in Florida. HEDGES, F.: *Sphæropsis tumefaciens* Nov. Sp., the cause of the lime and orange knot. *Phytopath.* **1**: 63–65. 1911. —— AND TENNY, L. S.: A knot of citrus trees caused by *Sphæropsis tumefaciens*. *U. S. Dept. Agr., Bur. Pl. Ind. Bul.* **247**: 1–74. 1912. RHOADES, A. S. AND DEBUSK, E. F.: Diseases of citrus in Florida. *Fla. Agr. Exp. Sta. Bul.* **229**: 104–106. 1931.

Diplodia disease of citrus (*Diplodia natalensis* Evans).—This disease causes injury in two different ways: (*a*) by a killing of the bark of branches of all sizes, accompanied by the oozing of gum; and (*b*) by causing a stem-end rot of the fruit similar to that caused by *Phomopsis citri*. Localized or extended lesions may be formed on large branches, or small branches may be killed back for some distance. The fruit rot can be distinguished from the Phomopsis rot by the darker color of the affected tissue. The disease was first studied in South Africa and has been reported from Cuba and Florida. EVANS, I. B. P.: *Transvaal Dept. Agr. Sci. Bul.* **4**: 1910. STEVENS, N. E. AND WILCOX, M. S.: The citrus stem-end rot "Diplodia"; its life history and relation to *Sphæropsis malorum*. *Phytopath.* **15**: 332–340. 1925. FAWCETT, S. H. AND LEE, H. A. *loc. cit.* pp. 409–412. 1926.

Chrysanthemum ray blight (*Ascochyta chrysanthemi* Stev.).—The blighting of the corolla of the ray flowers resulting in poorly developed, discolored, one-sided heads is the most characteristic feature of this disease. "If the case be severe, and a bud be attacked while still young, no rays will develop; the head will not open." Lesions may also be produced on the stems, and adjacent leaves may be involved. STEVENS, F. L.: Chrysanthemum ray blight. *Bot. Gaz.* **44**: 241–258. 1907. PAPE, H.: Blutenschaden bei Chrysanthemum. *Gartenw.* **31**: 604–606. 1927.

Stem rot and leaf spot of clematis (*Ascochyta clematidina* (Thüm.) Gloy.).—This disease may be evident as a stem rot alone or as a leaf spot and a stem rot. The

fungus may advance down the petiole into the stem and by its growth it may girdle and kill the stem, or independent internodal infections may have the same effect. Pycnidia may be produced on either leaf or stem lesions. GLOYER, W. O.: *Ascochyta clematidina*, the cause of stem rot and leaf spot of Clematis. *N. Y. (Geneva) Agr. Exp. Sta. Tech. Bul.* **44**: 1–14. 1915.

Hard-rot disease of gladiolus (*Septoria gladioli* Passer).—This disease is characterized by the production of lesions on the leaves, but those on the corms are of most concern. Seedlings and plants from cormels are more susceptible to leaf infection than older plants, the spots being reddish brown and more or less circular or with straight sides when the advance is limited by the midrib. Older lesions show light-gray centers with numerous pycnidia, surrounded by a zone of purplish brown to black. The final result on the corms is the production of sunken, dark or almost black spots of variable form and size with definite margins. The spots may be few or numerous and adjacent lesions may coalesce to form larger ones. The extreme effect may be the reduction of the corm to a hard, shriveled and wrinkled black mummy. Affected bulbs, if planted, may produce dwarfed plants which sometimes fail to blossom. MASSEY, L. M.: The hard-ot disease of the gladiolus. *Cornell Univ. Agr. Exp. Sta. Bul.* **380**: 151–181. 1916. GLOYER, W. O. AND CARPENTER, D. C.: Comparison of fungicides for the control of hard rot of gladiolus corms. *Bul. Am. Glad. Soc.* **4** (4): 20–22. 1927.

Tomato leaf spot (*Septoria lycopersici* Speg.).—While this disease is called the leaf spot, it also produces similar lesions on stems, calyx and flower pedicels and more rarely on young fruits. The circular, brownish or grayish leaf spots vary in size from that of a pinhead to several millimeters in diameter, and may be few in number or so numerous as to coalesce and completely blight the affected leaves. The heavy early defoliation which results in regions in which the disease prevails causes enormous annual losses due to reduction in yield and also to lowered quality of the fruit. The parasite has a narrow range of sporulation temperatures, *viz.*, 59 to 80.5°F., hence is unimportant except in certain parts of the Middle Atlantic and Middle Western states. The parasite lives over winter on dead leaves and vines of the tomato and can also survive on the dead remains of various crops. It can live as a parasite on various other species of Solanaceæ, such as common nightshade, horse nettle, ground cherry and jimson weed. Hence sanitary measures are of importance to supplement other control practices. LEVIN, EZRA: The leaf-spot disease of tomato. *Mich. Agr. Exp. Sta. Tech. Bul.* **25**: 7–51. 1916. COONS, G. H. AND LEVIN, EZRA: The leaf-spot disease of tomato. *Mich. Agr. Exp. Sta. Spec. Bul.* **81**: 1–15. 1917. MARTIN, W. H.: Studies on tomato leaf spot control. *N. J. Agr. Exp. Sta. Bul.* **345**: 1–42. 1920. MUNCIE, J. H.: Tomato leaf spot and experiments with its control. *Pa. Agr. Exp. Sta. Bul.* **177**: 3–23. 1922. PRITCHARD, F. J. AND PORTE, W. S.: The control of tomato leaf spot. *U. S. Dept. Agr. Bul.* **1288**: 1–18. 1924.

European canker of poplar (*Dothichiza populea* S. & B.).—This is an introduction from Europe which affects several species of poplars and cottonwood. Cankers are formed upon trunk or branches, followed by death of parts distal to the girdled zone. HEDGCOCK, G. G. AND HUNT, N. R.: *Dothichiza populea* in the U. S. *Mycologia* **8**: 300–308. 1916. DETMERS, FREDA: Dothichiza canker on Norway poplar. *Phytopath.* **13**: 245–247. 1923. HEDGCOCK, G. G.: *Dothichiza populea* and its mode of infection. *Phytopath.* **17**: 545–547. 1927.

CHAPTER XXIV

DISEASES DUE TO SMUT FUNGI

USTILAGINALES

The smut fungi have received their common name because of sooty-black spore masses or sori which are characteristic of many of the species. Not only are the spore masses generally black, but in a very large number of cases they break up into a fine dust-like powder, the individual spores or spore groups, which are readily dissipated by the wind. The smut fungi are all obligate parasites, although they produce a stage that develops independent of the host.

Hosts and Economic Importance.—The smut fungi attack many wild and cultivated hosts, but the species of most economic importance are those affecting the cereals—wheat, oats, barley, rye, corn, rice, sorghum varieties—and wild and cultivated grasses. A few other economic plants are also affected by smuts—for example, the onion, spinach and sunflower. The agricultural importance of the smut diseases is due (1) to the extensive and worldwide cultivation of the cereals; and (2) to the fact that the parts most frequently destroyed by the smuts are either the grains or the entire inflorescence, thus causing either "kernel" or "loose smut," although less frequently the foliage may be invaded, with the production of "flag smut." As a result of smut, heavy losses of cereal crops are experienced with marked reduction in yields and with a product of lowered quality.

General Characters.—The smut fungi cause either systemic or localized infection, that is, the mycelium of a smut pathogene may grow throughout the tissues of the plant from the seedling stage to maturity, or the mycelium may be localized in certain aerial parts. The characteristic features of the order are: (1) the formation of single or grouped resting spores (chlamydospores), which generally accumulate in black powdery masses or *sori;* (2) the germination of the resting spores to form either a *promycelium* (indirect germination), or an *infection thread or hypha* (direct germination); and (3) the budding of the secondary spores, or sporidia, in either soil moisture or nutrient solutions to form either yeast-like forms or secondary sporidia of a different type. In a few cases conidia are formed on the surface of affected parts, but this method of reproduction is uncommon (*e.g.*, *Entyloma spp.*).

In the indirect germination of the resting spores two types of *promycelia* are formed: (1) a two- to four-septate hypha with elliptical or oval, uninuclear *sporidia*, or secondary spores, which bud out from the distal

end of each cell (Ustilago type); or (2) a long or short continuous hypha which produces a crown or whorl of sporidia, or secondary spores, from its free end (Tilletia type). The sporidia may germinate to produce an infection thread or hypha which may infect the host, or these sporidia may bud indefinitely in nutrient solutions, and the buds give rise to infection hyphæ when conditions are favorable. In general, smut spores provided with a nutrient solution will make a much more copious growth than when germination occurs in soil moisture. Fusion of sporidia or hyphæ is fairly common in the smut fungi. In the direct germination, a resting spore forms a simple septate hypha, which may penetrate the host at once and establish an infection, or in a nutrient solution a much branched, septate mycelium may be formed.

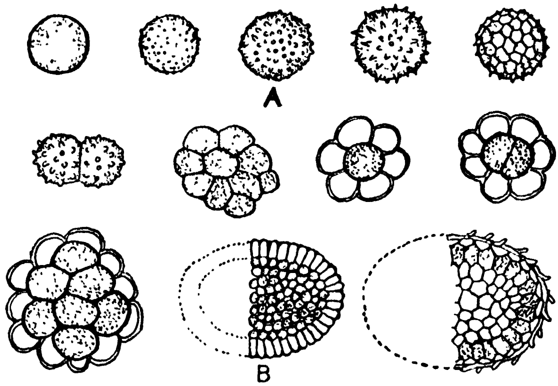

FIG. 207.—Various types of smut spores. *A*, single-celled spores, smooth and with various wall markings; *B*, series of spores from two to many celled. Stippled cells are fertile, others sterile.

Types of Infection.—Although the smuts show striking similarities in symptoms and effects, they exhibit important differences in the time and manner of infection. Three important types may be illustrated by the smuts of our cultivated crops:

1. *Seedling Infection.*—In this group of smuts, infection can take place only during the young or seedling stage, from spores that were either carried on the seed or were present in the soil or in loose smut of oats from mycelium that infected the glumes at flowering time. In seedling infection the addition of smut spores to the soil after seed germination has taken place will cause no infection, but if viable spores are present in the seed bed at seeding time, infection may result if moisture and temperature conditions are favorable. The bunt or stinking smut of wheat, the loose and covered smuts of oats, the covered smut of barley, kernel smut of sorghum and millet smut illustrate seedling infection from seed-borne spores. Soil contamination may result from *wind-blown smut*, as illustrated by the "smut showers" of the Inland Empire of the Pacific Northwest and some other similar sections, or it may be *residual*, that is, the

spores may originate from a smutty crop grown on the same ground the previous year or even at an earlier time. Residual soil contamination is an important feature of flag smut of wheat, onion smut and head smut of corn and sorghum. In seedling infection the infection threads which originated from either primary or secondary sporidia enter the young seedling, reach the growing point and then keep pace with the growth of the host tissues until the heads are formed, when the characteristic smut masses appear.

2. *Blossom Infection.*—In this type, which is illustrated by the loose smuts of wheat and barley, the infection takes place at blossoming time. The smut spores from smutted heads, which reach maturity about the time normal heads are in blossom, are blown by the wind, and by the direct method of germination infect young ovaries of normal heads, and produce an internal mycelium which is developed within the seed. When germination processes are started the mycelium keeps pace with the growing points of the young seedling, but does not reveal its presence until the heads are formed, when the spores are organized and matured. In this type the fungus is not carried over the winter in the form of spores lodged on the surface of the seed, but exists in the "germ," or embryo of the seed, as a vegetative mycelium, ready to resume activity with the awakening seed. It should be noted that infection takes place previous to the harvesting of the crop, and that infected seed, that is, seed in which an internal smut mycelium has been formed, if planted the next season, will produce plants affected with the loose smut.

3. *Shoot Infection.*—In this manner of infection, which is illustrated by the common smut of corn, infection is localized in various aerial parts, that is, there is no systemic infection as in the two other types. Spores may germinate on leaves, stalks or flower parts, and form infection threads which enter the host tissue and establish a mycelium. As a result of these local infections small or large smut masses or sori may be developed from the purely local mycelium, but there is no general spread of the mycelium within the host tissue from an original center of infection. Each smut sorus or smut boil that appears represents a separate infection. In shoot infection the primary or secondary sporidia are carried by the wind to the parts which they infect, and they may be residual in origin or the smut may be brought into the field from some other source.

The smuts are grouped in two families, which are characterized mainly by the method of germination and by the form of the promycelium.

I. Ustilaginaceæ

Germination is by means of a septate promycelium with lateral and terminal sporidia or by a septate infection thread which does not form sporidia. Sori generally form naked or covered, exposed, dusty or agglutinated masses, but in a few cases the spores may be buried within

the host tissue. The spores are continuous, two-celled or united in spore balls or groups. The principal genera of economic importance will be briefly characterized.

Ustilago.—Sori naked or without an enclosing membrane, and making black, dusty masses at maturity. Spores single, more or less globular, light to dark brown, smooth, finely verrucose or spiny. Germination direct or more frequently indirect.

Sphacelotheca.—Sori generally in the inflorescence, replacing the kernels, with external false membrane of fungous tissue and a central sterile mass of fungous tissue, a columella. Spores single, reddish brown, smooth.

Sorosporium.—Sori generally dusty, without enclosing membrane. Spores held in balls or groups by a mucilaginous matrix when young, but separating, or only loosely held together at maturity.

II. Tilletiaceæ

Germination is by a long or short, non-septate promycelium which forms an apical whorl of elongated or filiform sporidia. These sporidia may fuse in pairs or not and either form infection threads direct or produce secondary sporidia which are either similar or dissimilar. The sori are either dusty and exposed or permanently embedded in the host tissues. The spores are nearly hyaline, light brown or dark brown, single, with or without hyaline appendages and either dusty or not readily separating, or in groups or balls, the cells of the latter all fertile or with both fertile and sterile cells. The principal genera furnishing pathogenes of economic importance will be briefly described.

Tilletia.—Sori generally in the inflorescence replacing the kernels with "smut berries" or "smut balls," which are dusty when crushed. Spores single and smooth, spiny or reticulate. Sporidia filiform, generally fusing in H-shaped pairs.

Urocystis.—Sori usually in leaves or stems, forming dusty erumpent spore masses. The spores are compound, consisting of one to several dark, central fertile cells, surrounded by lighter-colored sterile cells. Promycelium with terminal fascicle of sporidia or forming hyphæ direct instead of sporidia.

Entyloma.—Sori generally in the leaves forming discolored spots, frequently light in color, hence the common name "white smuts." Aerial conidia may be formed on the surface of the spots, giving a white powdery appearance. The true smut spores are permanently embedded in the host tissue, single, hyaline or pale yellow to brownish, thick-walled, and frequently germinate *in situ*.

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BUNT OR STINKING SMUT OF WHEAT

Tilletia tritici (Bjerk.) Wint. and *T. levis* Kühn

This is a systemic disease of wheat which is due to seedling infection at the time of germination, but the injury does not become evident until the affected plants are in head, when the normal grains will be found

to be replaced by grains filled with a black powdery material, the smut dust or spores. It causes loss from reduction in both yield and quality.

The common name "bunt" is probably of English origin. It is supposed that affected heads were first designated as "burnt ears," with a later dialectic contraction to "bunt ear" and finally to "bunt." It is the pronounced odor of sour herring, produced by volatile substances given off from the smut masses, which has suggested the appropriate name of "stinking smut." There are two kinds of bunt, the smooth-spored form (*Tilletia levis*) being called "high smut," since the culms of affected plants are more often as high as those of normal plants, while in the rough-spored species (*T. tritici*), or "low smut," the culms of affected plants are frequently shorter than normal culms.

History.—Bunt or stinking smut of wheat was probably known in ancient times, but there was no word to express it in the language of either Greece or Italy. It was probably included under the general term of mildew or blight. Pliny speaks of the blasting of corn "which cometh of some distemper of the air," while Bacon says, "Mildew falleth upon corn and smutteth it." Stinking smut of wheat was apparently the first smut to attract attention and in 1733 Tull wrote "Smuttiness is when the grains of wheat, instead of flour, are full of a black stinking powder." In 1755 Tillet noted the difference between *la carie*, or stinking smut, and *le charbon*, or loose smut. As late as 1775 Bjerkander classified stinking smut as *Lycoperdon*, since the smut dust was supposed to resemble the spores of puff balls and was surrounded by a membrane thought to be similar to the peridium. The belief that smut was a fungus was not generally accepted at that time, and Tessier, in 1783, considered it as a degeneration of the grain and not the result of a parasite. This view that the smut spores were morbid eruptions of vegetable matter persisted for a long time, and various other notions were prevalent. Some even considered the smut an indication of an offended Deity. It is interesting to note that many erroneous notions have persisted even down to the present time among the uneducated farming population, since joint worms, poison, unfavorable weather conditions, etc. are proclaimed as the causal agents. In 1807 Prevost discovered that smut spores germinated in water and so were of the nature of fungi. These observations were confirmed by other observers and our knowledge was further extended by Tulasne in 1854. A few years later Kühn produced artificial infections and followed the penetration of infection threads into young seedlings, while Brefeld in 1883 gave more complete details of spore germination, especially in nutrient solutions. During following years the contributions on wheat smut became more numerous, and we now have a voluminous literature from writers in various parts of the world. During later years the emphasis has been placed upon seed disinfection as a means of control, as witnessed by numerous bulletins, and but few advances have been made concerning other phases of the subject. The relation of time of seeding and temperature to the per cent of smut in a crop was first definitely shown by the work of Munerati in 1911 and was confirmed by the work of Heald and Woolman published in 1915. That seed injury from bluestone treatments is greatly increased by the cracking of grain in threshing was noted by German workers as early as 1872, but was again brought to our attention in 1914 by Woolman. The failure of standard seed treatments to give control of bunt in the Pacific Northwest was shown by the researches of Heald and George to be due to the widespread wind dissemination of the spores, causing a general contamination of the summer fallow previous to seeding time. Previous to 1921 there had been no definite data available concerning the relation of the spore load of individual grains to the per cent of smut

appearing in the crop, but this information has been made available, so that it is now possible to make a microscopic examination of a wheat sample and predict the approximate amount of smut which may be expected under certain conditions if seed disinfection is not practiced.

Treatment of seed with copper sulphate to prevent bunt was first recorded by Schulthess in 1761, but Kühn in 1866 was the "first to investigate carefully the effect of copper sulphate on bunt spores as well as upon the seed and to recommend a definite formula." Although the hot-water treatment of Jensen (1888) was shown to be effective, and a few other chemical treatments were recommended, copper sulphate in some form continued to be the predominating seed disinfectant until the introduction of formaldehyde. This was suggested by Heuther in 1895 and soon came into general use in the United States following the publication of the work of Bolley in 1897. Formaldehyde largely replaced the use of bluestone, except in certain regions, like the Pacific Northwest, where its use was continued because of greater efficiency in preventing bunt due to a soil contamination. Both bluestone and formaldehyde, while effective, proved unsatisfactory because of seed injury, the latter especially in the semi-arid regions. The use of copper carbonate in dust form was first recommended by Darnell-Smith in Australia in 1917 and its use in America received special impetus from the work of Mackie and Briggs in California and Heald and Smith in Washington. The favorable results which have generally attended the use of copper-carbonate dust have resulted in a very general adoption of this treatment.

During recent years some emphasis has been placed on the use of certain organic mercury compounds, such as chlorophol, uspulun and germisan, as steeps, because of the elimination of seed injury and because of their stimulating effect upon germination and growth. The favorable reception of the copper carbonate dust treatment has led to the manufacture of organic mercury compounds, such as seed-o-san, semesan and Bayer's dust, for use as dusts. Uspulun and other organic mercury compounds have been more generally used in Germany than in the United States.

Geographic Distribution.—The stinking smuts of wheat are prevalent in greater or less extent in every country where that cereal is grown. Since the perfection and introduction of recognized methods of effective seed disinfection it has not produced such heavy loss except in certain sections like Australia or the Pacific Northwest. In the latter section, however, seed disinfection has not been effective for winter wheat. Bunt is rather uncommon in the warmer sections where wheat is grown—for example, in the southern United States or in the east, center and south of India. Its occurrence is more variable in the states of the central Mississippi Valley, where it may be rare one season and epiphytotic another in winter wheat. Epiphytotics have been especially noted in Nebraska and Kansas, due presumably to the abnormally low temperatures which prevailed at the time of seeding, giving optimum conditions for infection, while in average seasons in those localities the prevailing temperatures are too high for heavy infections to take place.

The rough-spored smut (*Tilletia tritici*) is the prevalent form in the Pacific Northwest and other wheat sections west of the Rockies, but the smooth-spored species is not uncommon, having been introduced since 1926. The rough-spored smut is not uncommon in some of the eastern states, although it is rarely found there as the prevailing form, the smooth-spored species (*T. levis*) generally predominating.

Bunt has continued to be a serious problem in the Pacific Northwest because of the peculiar combination of climatic factors and farming practices. The important contributing features are: (1) the very general system of summer fallow for winter wheat; (2) the exceptionally dry summers with little or no rain in July and August; (3) the progressive maturing of the wheat from the western or southwestern portion of the wheat area to the eastern portion; (4) the strong and prevailing winds which sweep from the regions of early harvest to the regions of late harvest; (5) the general

and widespread dissemination of the smut spores (smut shower) from the various threshing operations, resulting in a heavy soil contamination of the summer-fallow seed beds; and (6) the moderate temperatures which prevail at the time when the most of the fields of winter wheat are seeded.

Symptoms and Effects.—Bunt of wheat does not become evident in a crop until the wheat is in head, since it is only the inflorescence that shows the pronounced deviation from normal. A microscopic examination a few days prior to the emergence of the head from the boot shows, however, striking differences in normal and smutted florets. The pistils from smutted heads are larger with an ovary double the length of a normal healthy ovary and green instead of almost white. The stamens from smutted heads are reduced in length and breadth, the anthers are pale yellow instead of green, much reduced in size and without perfectly organized pollen cells.

After the emergence of the heads the presence of smut can be easily detected, since it causes cetain deviations from the normal in the form of the infected heads. This is soon evident in the "compactum" varieties, such as Little Club, Hybrid 128 and others, since the normally compact or square heads are generally changed to a more slender type and may in some varieties exceed the healthy heads in length. In the "vulgare" types the deviation from normal is less striking, and does not become evident until the smutted berries begin to expand and so cause a divergence of the glumes and thus give the head a more loose or open appearance. It is generally true that smutted heads previous to maturity exhibit a darker green color than normal heads, and also remain green longer. This condition is especially noticeable in such varieties as Winter Fife and Bluestem. When the wheat is in the "dough" stage, a verification of the presence of smut may be easily made by pinching the grains with the thumb and forefinger, the smutted grains showing at this time that they are filled with a soft, black, pasty mass. In mature grain the same method may be necessary in examining "vulgare" types when accurate quantitative counts of smutted heads are being made. At this time the black, pasty interior has changed to an oily powder, the characteristic smut mass. In some varieties the infected heads stand erect, when normal ones are beginning to droop as a result of the increasing weight of the ripening grain. In bearded varieties, smutting frequently causes a shedding of the awns with the approach of maturity. The presence of smut in a field can frequently be detected by the characteristic odor, similar to that of sour herring.

Since this disease is caused by an internal parasite, it is natural to expect certain responses to its presence. It should be noted first that the smut fungus is living at the expense of its host plant, the wheat. Its effect on the host may be summarized as follows: (1) the consumption of food; (2) the destruction of seed in the sporulating process; (3) the stimulating or retarding effect on normal physiological processes.

Badly smutted plants remain in many cases undersize and produce smaller heads than normal plants with increased tillering. The effect is the same on both species but is less pronounced on *T. levis*. Observations and experiments lead to the belief that stools may harbor the smut fungus, when no smut develops in the heads. In such cases the mycelium,

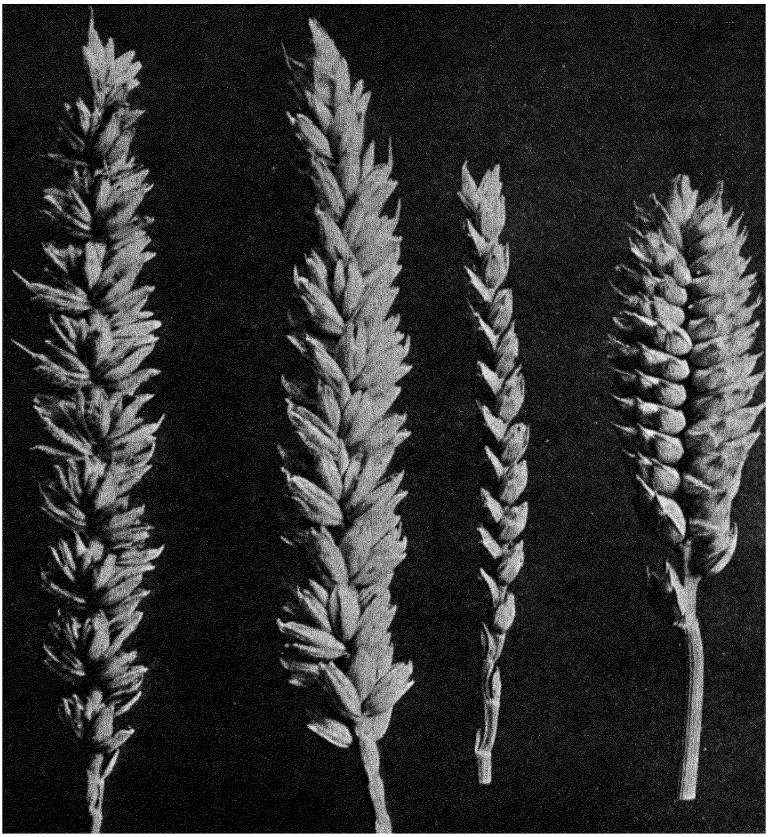

FIG. 208.—Smutted and normal heads of Jones Winter Fife and Hybrid 128. Note the marked change of form of the smutted head of the club wheat.

or vegetative body of the fungus, fails for some reason to reach the heads. This has been called invisible or "latent" infection (Gieseke, 1929; Zade, 1931), in contrast with normal or "open" infection, and as reported results in shortening of the culms and in reduced yield over uninfected controls. This behavior is substantiated by the observations that infection takes place with equal facility in both resistant and susceptible varieties, the further advance of the parasite being dependent upon the degree of susceptibility (Woolman, 1930). The condition might be expressed in this way: In a young infected seedling there is a race between

the smut fungus and the growing points of the flowering shoots in the upward growth. In some cases the fungus falls behind and never enters the heads, while in others it reaches its goal and penetrates the ovaries.

The most evident injury from wheat smut is due to the destruction of the grain or berry in the production of spores. The smut fungus enters the young ovary and uses up the food that is ordinarily accumulated and at the same time destroys the embryo, so that a fully smutted grain consists of only the brown outer seed layer (pericarp) enclosing the mass of smut spores.

A plant may be wholly or partially smutted, that is, all heads produced by a given stool may be smutted or only part of them may be invaded. The completeness of smutting varies with the different varieties and with the same variety fluctuates to some extent, apparently being influenced by the conditions which prevail during development. In general, the more resistant the variety the greater the number of partially smutted plants and partially smutted heads. A smutted plant frequently produces heads which are only partially smutted, that is, some grains may be normal, while others are infected. The normal grains may be variously distributed, bearing no definite relation to position. Partially smutted grains are sometimes very common and the degree of smutting varies from those which show a minute black speck to those in which nearly the entire grain is involved. Heads have been found which showed only a single partially smutted "berry," all the others being uninfected. The question is naturally suggested at this point as to whether there may not be an invisible infection, since there are all degrees of visible infection. The production of partially smutted berries is much more common in some fields than in others, and suggests a possible explanation for some of the ineffective results of seed treatment, but up to the present all attempts to germinate partially smutted berries have failed.

Losses from Bunt.—This is one of the most serious diseases of wheat, since it causes a complete destruction of the affected berries or grains, the agricultural product of the crop. The injury or financial loss to be charged to the bunt account is fourfold: (1) increased cost of production due to seed treatment, soil sanitation and cultural practices designed to reduce infection; (2) the reduction in yield per acre; (3) the lowering of grade or quality; and (4) the losses from separator and grain fires caused by smut explosions.

Seed treatment involves much extra labor, the use of enormous quantities of fungicides, the use of an increased amount of seed per acre due to reduced germination of treated seed and occasional complete failures, with the necessity of reseeding due to the killing effect of fungicides employed. The most pronounced seed injury has occurred in the dry sections where formaldehyde-treated wheat has been seeded in the dust, but the injury has been more constant in the regions which have retained

the bluestone treatments. This will be still further discussed under Seed Treatment as a method of control.

The amount of smut in a field or the per cent of smutted heads is generally taken as an index of the reduction in yield or loss from the disease, that is, with 10 per cent of all heads smutted, the loss would be estimated at 10 per cent. It seems probable, however, that the reduction in yield is not quite equal to the per cent indicated by the smutted heads, since smutted plants are generally weaker than the adjacent healthy plants, which consequently stool more heavily and tend to occupy the space. With higher per cents of smut the loss or reduction in yield is more nearly equal to the per cent of smutted heads, but it seems probable that there is little actual reduction in yield when the per cent of smutted heads is five or less. In regions in which soil contamination is not a factor, there should be no smut, or only traces if some standard seed treatment is practiced. The heavy smutting occurs either when there is no seed treatment or where there is a soil contamination. The maximum amount of smut recorded for a farm field in some extensive surveys of the Inland Empire was 88 per cent. In this case the seed had not been treated. Throughout the Pacific Northwest where summer fallow is the regular practice it is not uncommon to find 20 to 30 per cent of smutted heads in fields of winter wheat which have been seeded with carefully treated grain. It may be noted that seed of a susceptible variety which has been artificially coated with smut powder, so that it is carrying its maximum load of spores, when planted under the most favorable conditions will frequently produce 90 to 99 per cent of smutted heads.

When grain from a smutty field is threshed, many of the smut balls break and the black spore powder is distributed over the surface of the normal grains, lodging especially in the "brush" or tuft of hairs opposite the germ end. Grain from badly smutted fields is therefore conspicuously blackened and its value for milling purposes is correspondingly lessened, since special scouring machinery must be used to clean it. Conspicuously smutted wheat is also less desirable for feed, especially for poultry, hogs and sheep, since it is reputed to induce digestive disturbances. The lowering in quality is not always proportional to the amount of smut in the field, since maturity and moisture at the time of cutting and threshing, as well as the variety, affect the breaking of the smut balls. The farmer who produces smutty grain suffers a dockage in price in accordance with the amount of smut, and it is specified by the U. S. Grain Standards Act that smutty wheat must be so designated when offered on the market.

When smutty wheat is threshed, explosions and fires may result from the formation of an explosive mixture of dust and air which is ignited by static electricity developed during the threshing operations. Explosions are favored by extremely dry conditions, which increase the quantity of

dust produced from both smut and straw. Smut fires and explosions are most frequent in the arid or semiarid regions of the Pacific Northwest, although they have been of occasional occurrence in regions east of the Rocky Mountains. The losses from smut explosions are due to damage to separators and to grain fires. The damage to separators varies from slight to total destruction. In the investigations reported by the U. S. Department of Agriculture for the Pacific Northwest, the average percentage of smut in 108 explosions was 15. It was also shown that in 146 explosions and fires nearly 30 per cent of the separators were a complete loss. Added losses are due in many cases to the fact that the flames spread into the straw pile and ranks of sacked grain and also to the unthreshed grain in the field. These losses are, however, being reduced as a result of safety devices to prevent explosions or to extinguish fires.

Etiology.—Bunt of wheat is due to either *Tilletia tritici* (Bjerk.) Wint., the rough-spored smut, or to *T. levis* Kühn (*fœtans*), the smooth-spored smut, two very closely related species of the Tilletiaceæ having nearly an identical life history. The spores of *T. tritici* are spherical or nearly so, 15 to 20μ, occasionally oblong to pear shaped, reaching a maximum length of 22μ. The epispore is marked with ridges which form a network with meshes, variable in size and shape, generally 3 to 4μ wide. The spores of *T. levis* are globose to elliptic, occasionally somewhat angular, very variable in form and size, 16 to 18μ or 19 to 25 by 16 to 17μ and have a smooth epispore.

The first proof of the infective character of the smut dust was offered by Tillet in 1755, by sowing clean and artificially smutted seed in adjacent rows, although he did not at that time recognize smut as either a parasite or a fungus. The rough-spored smut was first named *Lycoperdon tritici* in 1775 by Bjerkander, and *Tilletia caries* by Tulasne in 1847. The smooth-spored species was first named *T. fœtans* by Berkeley in 1860, but no description was published until that of Kühn in 1873, when the present name, *T. levis*, was given.

Infections of bunt occur only during the young or seedling stage under normal field conditions. Artificial infections of plants may be made previous to flowering, by applying the inoculum to culms broken off near the base (Milan, 1928) by hypodermic injections and by inserting the inoculum inside the leaf sheath with a transfer needle (Bodine and Durrell, 1930). When living spores are introduced into the soil along with the seed wheat or exist in the soil in which wheat is planted, smut will appear in the crop if proper conditions have prevailed during the germination period. The smut spore germinates by the production of a hypha-like growth, the *promycelium*, which produces at its free end a fascicle of four to 12 long, narrow, curved spores or *sporidia* frequently united into H-shaped pairs. Infection threads may arise from these sporidia, or secondary sickle-shaped sporidia may be formed which later

develop infection threads. Under favorable conditions one or more infection threads (there is evidence of multiple infection) penetrate the young seedling and reach the growing point of the shoot. Here the fungous threads or hyphæ keep pace with the growth of the host, but give little or no external evidence of their presence until the production of heads, when they enter the ovaries and begin the development of the spores which reach maturity at or slightly before harvest time. The

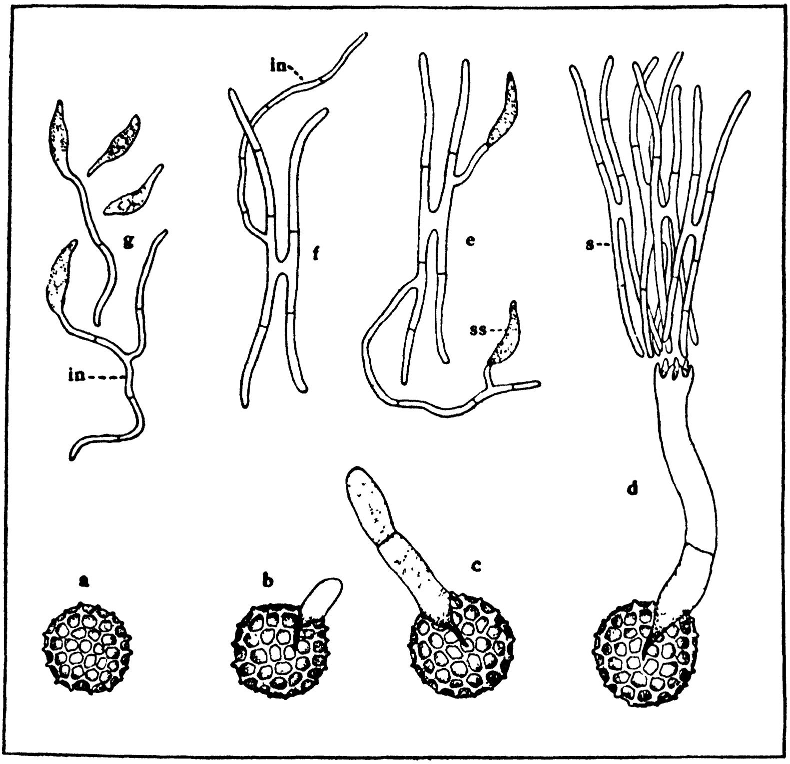

FIG. 209.—Various stages in the germination of spores of stinking smut (*Tilletia tritici*). *a*, spore surface showing characteristic reticulate ridges; *b*, *c*, stages in the formation of the promycelium; *d*, mature promycelium with terminal fascicle (*s*) of H-shaped sporidia; *e*, a separated sporidium which has germinated to form secondary sporidia (*ss*); *f*, a separated sporidium which has given rise to an infection thread (*in*); several secondary sporidia which have started to germinate and have produced infection threads.

general plan of the life history as outlined is typical of the various smut species in which a *seedling infection* occurs.

The smut "balls" or "berries" produced by infected plants vary in size and each one contains from six to nine million spores. During the threshing operations a certain per cent of these smut balls will break and the smut dust will be carried out through the stacker, or many will lodge upon the surface of normal grains—the groove, or suture, and the brush, or tuft of hairs at the terminal end, serving as resting places for

large numbers. Normal wheat grains from an infected field may have so many spores lodged on their surfaces as to give them a dark color, but other grains which show no smut to the naked eye may still be carrying a sufficient number of spores to produce a smutty crop. Smutted wheat may also contain unbroken smut balls and partially smutted grains, which are an added source of danger. Experimental tests show that there is but little danger that the smut balls that have passed through the threshing machine without breaking will rupture during treating and seeding, especially in moderately smutted wheat. The idea that the unbroken smut balls are a source of danger has been based more on theory than on practice. Seed from smut-free fields, if obtained from regions in which a smut shower occurs, may be carrying smut which was blown onto the standing grain or lodged on the shocks after cutting.

Even if smut-free seed is used, there are two other possible sources of infection: first, from soil contamination from a previous smutty crop on the same ground; second, soil contamination from wind-blown spores. If wheat follows wheat and the first crop was smutted, large numbers of smut heads left in the field may be a source of danger. Free smut dust in the moist soil will lose its infective properties after 50 to 60 days (3 to 4 weeks according to Bonne, 1931) and will not survive winter temperatures, but spores in heads or unbroken balls may retain their virulence and viability for a year or more under field conditions. The wind-blown smut is, however, the most important source of soil contamination, especially in regions in which summer fallow is practiced. In the Palouse country the "smut shower" begins in August, reaches its maximum about the middle of September and then gradually subsides, but the total spore fall frequently amounts to enough to make an average of over five million spores for each square foot of exposed summer fallow. Seedings in summer fallow in this region just preceding, during or immediately after the climax of the spore fall, are likely to be heavily smutted even though clean or carefully treated seed is employed.

Predisposing Factors.—The amount of smut produced by a given seeding will, of course, depend upon the spore load, that is, upon the average number of spores carried by each grain of wheat and upon its viability, but the most important environmental factors are the amount of moisture in the soil and the temperature during the period of germination. The minimum percentage of smut is produced in very dry soil, the maximum in a soil with moderate moisture, but infection is reduced by seeding in a very wet soil (Hungerford, 1922; Caspar, 1926; Rabien, 1927). This behavior will explain some results of farm experience. The deficiency of oxygen inhibits spore germination in a very wet soil.

The time of sowing, for instance, exercises a great influence upon the strength of attack. Numerous experiments and observations have been published, and the general result is that the temperature at which the germination of the spore

and grain takes place is the most important factor. The wheat must, therefore, germinate faster than the spore, in order to evade attack. This may be possible with higher as well as lower temperatures, which thus demonstrate the fact that minimum, optimum and maximum temperature are different with respect to the wheat and the smut.

The following Centigrade temperatures for germination have been recorded:

	Wheat	Smut	
		Heuser, W.	Woolman, Humphrey
Minimum	3–4 5	5	0–1
Optimum	25	16–18	18–20
Maximum	30	25	25–29.1

Slight variations in these figures have been reported by later workers (Hahne, 1925; Rabien, 1927; Bonne, 1931), but not sufficient to alter the behavior.

A fast germination is altogether an advantage in wheat. When the wheat germinates at very high temperatures, 20 to 25°C., it then approaches the optimum for its germination, while the spore has passed its optimum. The wheat will thus germinate faster than the spore, and has got a good start ahead of the spore at the time of the infection state (Heuser-Holm).

It may be noted that some regions are exceptionally free from smut (east, center and south India; southern Russia, the Spokane Valley in Washington), and it has been pointed out that this is due to the high soil temperatures which prevail at the time of seeding operations, thus insuring a rapid germination of the wheat. The results obtained at Pullman, Wash., in 1916, as shown in the following series of periodic seedings of carefully treated Hybrid 143 on summer fallow, are typical of the conditions in eastern Washington.

	Per Cent Smut		Per Cent Smut
Aug. 11	0.00	Sept. 25	30.98
Aug. 24	1.77	Oct. 2	41.14
Sept. 1	12.85	Oct. 6	16.92
Sept. 8	18.85	Oct. 10	15.08
Sept. 11	14.22	Oct. 18	11.49
Sept. 21	38.07	Oct. 30	Winter killed

The low per cent of smut in the early seeding was due to two factors: first, the relatively high soil temperatures; second, the fact that the spore fall in August had not seriously contaminated the summer fallow. The reduction of smut during the late seedings is explained by the gradual loss

of infective power of the smut spores and the unfavorable temperature conditions.

Physiologic Strains.—The definite recognition of physiologic strains of bunt is a comparatively recent development. Previous to 1924 when the first evidence of physiologic specialization was published (Faris), it was generally believed that physiologic strains of bunt did not exist. By the use of differential hosts Rodenhiser and Stakman (1927) showed the existence of three forms of *T. levis* and two of *T. tritici*, using collections from Minnesota and several foreign countries. Reed (1928) by similar methods obtained four races of *T. levis* and six of *T. tritici*. Roemer (1928) showed that physiologic strains exist, but he did not attempt to determine the number represented by his collections. In his first report (1928) Gaines reported three strains of *T. tritici* and two of *T. levis*, but in 1930 (Heald and Gaines) the number of *T. levis* strains had been increased to four, making a total of seven strains. Reichert (1930), working in Palestine, obtained six different strains of *T. tritici*. The existence of numerous physiologic strains has now been definitely proved, one to several being prevalent in a given locality. The appearance of new physiologic strains or their introduction from some other locality is the explanation for epidemics of bunt in varieties formerly thought to be very resistant, as, for example, the recent epidemic on Durum wheat in Minnesota (Holton, 1930, 1931). Up to date it has not been possible to separate the strains by characters shown in artificial cultures but only by their reaction on selected differential hosts. Flor (1932) has been successful in hybridizing *T. levis* and *T. tritici* and also the various strains within the species, which lends support to the belief that new physiologic forms may appear in nature at any time. It is of interest to note that Bonne (1931) found that *T. tritici* from different varieties of the same origin showed equally as great differences in capacity to infect various varieties as did collections from different and widely separated localities. The numerous physiologic strains certainly complicate the work of breeding for resistance.

Host Relations.—The various species of Triticum have been tested for resistance and have been found to show wide variation among the species and also of the varieties within the species. They have varied from complete freedom from smut, possible immunity, to nearly 100 per cent of infection. Susceptibility is found within all the following: common wheat (*Triticum vulgare*), club or square-head wheat (*T. compactum*), poulard or English wheat (*T. turgidum*), durum wheat (*T. durum*), Polish wheat (*T. polonicum*), einkorn (*T. monococcum*), spelt (*T. spelta*) and emmer (*T. dicoccum*). Recently Gaines and Stevenson (1923) have reported *T. tritici* on winter rye and on rye-wheat hybrids which have all of the morphological characters of a true rye. Smut is of principal concern on the true wheats. The "compactum" types are generally very

susceptible, the "vulgare" types variable, while durum wheat has been reported as hard to infect. Von Tubeuf suggested that resistance is correlated with the quick germination and rapid growth of the seedlings, and this varietal behavior has been studied by numerous workers, but there is little evidence that resistance can be explained in this way. Resistance does not appear to be connected with anatomical characters, but rather with chemical peculiarities of the host, showing different kinds composed of multiple factors in the various varieties, and definitely heritable. The most important work dealing with the susceptibility of wheat varieties has been carried out in Germany, Australia and in the United States. Some very resistant varieties have been found in all countries and it is noteworthy that some of these would be entirely smut-free during certain years. Many reports on varietal susceptibility have been published, but these are largely invalidated by our recent discoveries of physiologic strains of smut. Under a given environment different varieties of true wheats will vary from immunity or high resistance to 100 per cent infection.

Seed of the same varieties from different sources when tested side by side and given equal opportunity to develop smut may show great variation in the percentage of infection. For example, seven strains of Turkey showed 14.28 to 64.92, while six strains of Marquis varied from 18.39 to 42.55 per cent of smut.

In tests reported in 1925 (Tisdale, *et al.*), including a large number of varieties, Martin and Hussar were reported immune, but later work (Heald and Gaines, 1930) has shown these varieties to be immune only to certain physiologic strains, but highly susceptible to others. The same behavior has been shown for Albit, a promising new hybrid. Ridit, a Florence-Turkey (Washington) hybrid, has shown a rather high degree of resistance, being immune to some strains and only moderately infected by others. Some varieties of wheat appear to be highly susceptible to all of the physiologic strains of bunt—for example, Hybrid 128.

Three types of resistance (Gaines and Singleton, 1926) have been recognized: (1) resistant when fall seeded but immune when spring sown (Turkey); (2) resistance the same when seeded in either spring or fall; and (3) susceptible when fall seeded but highly resistant when spring seeded (Hope, Martin). Hope when fall seeded showed a variation from 40 to 78 per cent of smut when inoculated with seven physiologic strains but was entirely immune to the same physiologic strains when spring seeded (Heald and Gaines, 1930).

Prevention or Control.—In the past most emphasis has been placed on seed disinfection, but it must be evident that bunt will not be controlled by any single practice, but rather by the combined use of various methods. Consideration should be given to five different lines of attack as follows:

1. *Crop Rotation.*—Wheat following wheat is very likely to be smutty because of contamination of the soil from smut produced by a previous crop. Even with an intervening summer fallow, the smut from a previous crop may be a source of infection. The rotations must be suited to the conditions which prevail in the various sections.

2. *The Use of Clean Seed.*—Under uniform conditions, the amount of smut produced will depend upon the spore load, that is, upon the average amount of smut carried by each grain. Wheat that is *visibly* smutted may carry from 5000 to 263,000 smut spores per grain, and will produce a moderately or heavily smutted crop. Wheat that is visibly clean may carry up to 5000 smut spores per grain and may be expected to yield to 10 to 15 per cent of smut, according to variety and the spore load. It is possible by a microscopic analysis to predict the approximate amount of smut which a given sample will produce under optimum conditions. Visibly smutty seed should not be used when it is possible to obtain smut-free or visibly clean seed. If wheat showing unbroken smut balls must be used for seed, it should be thoroughly cleaned to remove as many of the smut balls as possible. The advantage of cleaning the wheat is further emphasized by the fact that the small grains produce more smut than the large plump grains.

3. *Seed Disinfection.*—Seed treatment should be practiced with some standard fungicide unless the spore load is below the danger point or seeding is carried out under conditions unfavorable for infection. It should give practically perfect protection except where there is a soil contamination. The following fungicides have given effective control of seed-borne smut in farm practice:

a. Copper sulphate or bluestone, 1 pound to 5 to 10 gallons of water for 5 to 10 minutes, with maximum time for the weaker strength (1–10 formula). The addition of salt recommended by some workers does not increase the effectiveness (Bodnar *et al.*, 1930).

b. Bordeaux, 4–4–40 or 8–8–40 formula, for 10 to 15 minutes.

c. Formaldehyde, 1 pint or pound to 40 gallons of water (1–320) for 10 minutes.

d. Uspulun or germisan, 0.25 per cent solution for 30 minutes.

e. Copper carbonate dust 2 to 3 ounces per bushel.

f. Organic-mercury dusts (*e.g.*, ceresan) 2 to 3 ounces per bushel. While the organic-mercury preparations are effective both in liquid and in dust form, their higher cost limits their use. The use of mercury dusts is forbidden in France because of their toxicity to man (Arnaud and Gaudineau, 1930).

Recently the effectiveness of copper oxychloride has been emphasized (Petit, 1928; Arnaud and Gaudineau, 1930).

The steeping in the liquid fungicides will give the better results by the open-tank or sack methods, than by the sprinkling method. The blue-

stone and the copper carbonate treatments will give some protection against infection from soil contaminations, but formaldehyde is of no value in such cases. Bluestone and formaldehyde treatments cause more or less reduction in germination. This may be greatly reduced by either presoaking (10 minutes in water, followed by 6 to 12 hours in wet sacks) or by following the dipping with a milk of lime bath (1 pound lime to 10 gallons of water for 5 minutes). Equally good results can be obtained by washing the formaldehyde-treated seed in water. Neither the copper-carbonate dust nor the organic mercury compounds cause any seed injury. It has been shown that most copper salts do not kill bunt spores in the steeping treatment, since the copper ions are only adsorbed, as is shown by subsequent germination if the copper is removed. Possibly some failures of copper treatments may be due to the removal of the copper in the soil (Hollrung, 1925; Bodnar *et al.*, 1927, 1930). Copper ammonium sulphate kills the spores since the complex ion in this compound penetrates into their interior.

The important advantages of the copper carbonate dust are as follows: (*a*) the elimination of the inconvenient and disagreeable soaking methods; (*b*) the convenience of being able to treat seed days or even months before it is needed for use; (*c*) the elimination of seed injury and retarded growth, with even an improvement in germination over the untreated seed; (*d*) the elimination of danger from seeding in the dry or in the dust (the cause of heavy losses with formaldehyde-treated seed); (*e*) increased yields due to no retarding influence of the fungicide, and better stands, with smut control equal to or better than that obtained by the other methods. Copper-carbonate dust treatment is not always equally effective. Best results are obtained if the soil is only moderately moist during the germination period. For this reason it is claimed to be unsatisfactory for regions having heavy rainfall (Volk, 1927). Under moist conditions copper carbonate-treated grain, if left in the drill, may set and cause breakage when the drill is started (Leukel, 1930). Numerous types of seed-treating machines are on the market for making the liquid treatment, and dusting machines have been described by Heald and Smith (1922), Mackie and Briggs (1923), and others, and a number of companies are now producing continuous rotary dusters.

4. *Cultural Practices.*—The following demonstrated facts should be kept in mind: (*a*) Seeding in relatively dry soil gives less smut than seeding with abundant moisture; (*b*) deep seeding causes more smut than shallow seeding; (*c*) early seeding either before the smut shower or at least before the fall rains begin will give either a smut-free crop or a low per cent of infection; (*d*) seeding during periods of relatively high temperatures will reduce infection; (*e*) seeding of summer fallow during the first few weeks following rain will generally result in much smut; (*f*) replowing of summer fallow reduces the amount of smut when there

is a wind-borne source of contamination; (*g*) with clean treated seed a fall stubble crop will show less smut than an ordinary summer fallow in regions where smut showers occur; (*h*) late fall planting will tend to decrease the amount of smut; (*i*) separated spores (smut dust) never live through the winter period under normal conditions, hence wind-borne spores are of no consequence in spring varieties.

5. *Selection of Resistant Varieties and Breeding for Smut Resistance.*—First consideration in any environment must be given to varieties which will show high producing power. Some very susceptible varieties, for example, Hybrid 128 under eastern Washington conditions, may be more profitable than some more resistant varieties. Careful attention to the testing of varieties will probably show resistant high producers adapted to the various sections. The ultimate aim of the plant breeder is the production of immune varieties of high producing capacity and prime quality. This is a task of some magnitude, since smut-immune varieties adapted to one locality may be poorly suited to another and protection must be provided against various physiologic strains. At present the experience of the local experiment stations should guide the growers in the selection of varieties best suited to their environment.

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LOOSE SMUT OF WHEAT

Ustilago tritici (Pers.) Jens.

This smut, peculiar to the wheat plant, is characterized by the complete destruction of the spikelets of affected heads, which become trans-

formed into black, powdery smut masses that are dissipated by the rain and wind previous to harvest time. The common name of *loose smut* will serve to distinguish the trouble from *bunt or stinking smut,* which is really a kernel smut, and the *flag smut* which forms the smut powder on the leaves and culms. In Australia the heads destroyed by loose smut are sometimes called "snuffy ears" by the farmers.

History and Geographic Distribution.—Since the three common loose smuts of cereals, wheat, barley and oats, have a very similar external appearance, it was natural that early botanists should consider them identical. They were first known under the name *Ustilago segetum* Bull., and the loose smut of wheat was named *Uredo tritici* in 1801, by Persoon. The name Ustilago was first applied to the loose smuts in 1552 by Tragus. *Ustilago carbo* and *Uredo carbo* were also in use before the loose smuts were known to be distinct species. As late as 1889 Plowright considered all of the loose smuts under the name of *Ustilago segetum.* About this time Jensen of Copenhagen (1888) was carrying out his work on the biology of these loose smuts and came to the conclusion that the two forms on barley which he called *tecta* and *nuda* were distinct, and he showed definitely that the loose smut of wheat could not infect any of the other cereals. Even after the loose smuts of barley and wheat were shown to be distinct, they were thought by some to be biological strains of a single species.

The true nature of the loose smuts of barley and wheat was not understood until some years later when Maddox of Tasmania (1895–1897) produced the first evidence of "blossom or intraseminal infection." A year later, according to Hori, the results of Maddox were confirmed by Yamada (1896) and then by Nakagawa (1898), a Japanese worker, who made field inoculations, introducing the spores into the flowers of the wheat with a forceps, but no histological details of the infection were worked out. The work of these pioneers was largely overlooked until the researches of Brefeld (1903) and Hecke (1904) again demonstrated the blossom infection in both species, and substantiated this by histological studies showing the mycelium of the pathogene in the growing points.

Seed disinfection practices showed that the life history of the loose smut of wheat was different from that of bunt or stinking smut. Plowright (1889) recorded the fact that the disinfection of wheat seed with copper sulphate for bunt control, as practiced on every well-managed farm, had no effect on the loose smut. This was later explained by the intraseminal mycelium which was out of reach of the fungicides, and the modified hot-water treatment which was devised by Jensen (1887–1889) overcame this difficulty. His first work was done with the loose smut of barley. The modified hot-water treatment was recommended for wheat by Swingle in 1894 and by Jensen in 1895, and since that time has been tested and recommended by various workers. Special mention should be made of the work of Freeman and Johnson (1909), Gregory (1923), Tapke (1924, 1926, 1929), Tiemann (1925) and Grevel (1930).

Loose smut of wheat is found wherever wheat is grown, but in many environments it is not sufficiently abundant to be a factor of importance in wheat production. For example, in northern India the amount does not generally exceed a fraction of 1 per cent, while in the Central Provinces 10 per cent is sometimes reported. It is so rare in the Inland Empire of the Pacific Northwest that it is practically unknown to the rancher, but it can generally be found by a search in any field, the amount only rarely reaching as high as 1 per cent. In some of the wheat-producing sections of the United States east of the Rockies it is sufficiently prevalent to call for control measures.

Symptoms and Effects.—This smut appears in the field shortly before the normal plants are in head and the spikelets of affected heads are

transformed into black, powdery structures which can be detected even before emergence from the boot. The smut masses are at first covered by a delicate grayish membrane, but this soon bursts and exposes the smut dust, which is gradually liberated and washed down by rains or blown away by the wind. The general rule is for every spikelet of a head to be smutted, but partially smutted heads may be found. In such cases it is always some of the upper spikelets that are left intact. All of the heads of a stool may be smutted or only part of them, but this

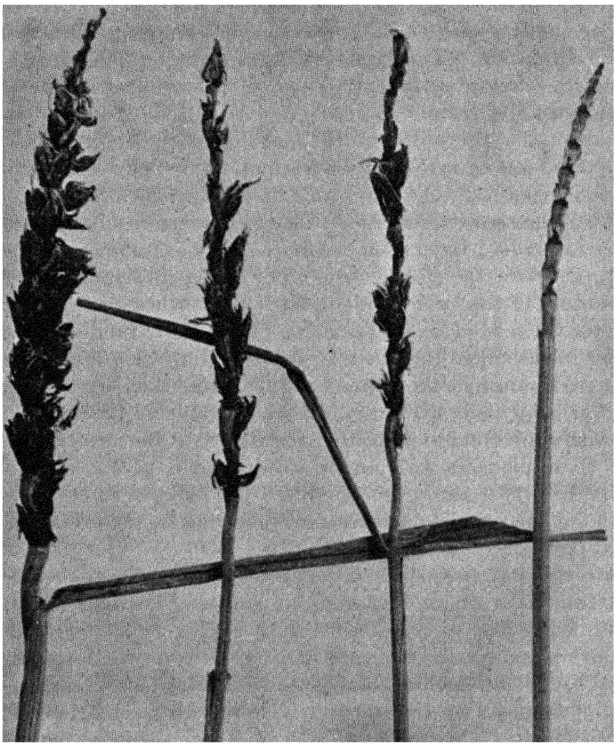

FIG. 210.—*Ustilago tritici*, loose smut of wheat.

behavior seems to vary with location and varieties. McAlpine (1910) states that it is uncommon to find shoots with both smutted and sound heads. In smutted heads the affected spikelets are completely destroyed, the only parts which escape transformation being the tips of awns in the bearded varieties. The central axis or rachis is never destroyed, but will finally be left as a bare structure with a few remnants of the spore masses. Smut masses may sometimes appear on the leaf blade, leaf sheath or culm. This has been observed in Australia, Egypt, India and Germany (Riehm, 1914; Tiemann, 1925). According to McAlpine (1910), "when a stool is affected with loose smut, the stalks are generally of a purplish tint, so that they can be readily picked out from among the

general crop." The same has been observed in the naked smut of barley, but is not particularly so in the case of the loose smut of oats. The purplish color is natural to some wheats, such as Purplestraw, but it occurs in other varieties when smutted.

The smut masses have reached maturity and the spores are being scattered by the wind by the time normal heads are in flower, and by harvest time the smutted heads are very inconspicuous, since the smut mass has been entirely dissipated, leaving the bare stalks. The covering membrane generally bursts before the smut mass is pushed out of the boot, but sometimes the smut heads remain hard and black, without becoming powdery, and the spores are not set free. The amount of smut varies under field conditions from a trace to as high as 15 to 25 per cent, depending upon the location and the variety. The highest per cent of smut in Kansas in 1906 occurred in two Japanese varieties (6 and 9 per cent), a Roumanian wheat (8 per cent) and a Kansas hybrid (15 per cent). Brentzel (1926) reported 3 to 25 per cent on Kota in North Dakota. It may be noted from these figures that the per cent of infection does not reach as high as in the bunt or stinking smut of wheat.

It is of interest to note that infection with the loose smut has a pronounced effect on physiological processes. Transpiration of smutted plants is 20 to 23 per cent more than that of normal plants. Smutted plants make a more active growth than normal ones for the first 20 to 25 days, but after that growth declines and by flowering time the dry weight of smutted plants is only 60 to 64 per cent that of normal plants (Kourssanow, 1928). Infection appears to increase winter killing (Tapke, 1929).

Since the chaff and the grain are completely destroyed and the smut scattered before harvest, the effect of the smut is different from that of bunt or stinking smut. The loss must be based on the reduction in yield alone, since quality is not affected unless one is considering the value for seed purposes. The product of the normal heads of a smutty field appears perfectly normal, even though infection has occurred. It is customary to figure the reduction in yield equivalent to the per cent of smutted heads, but in low degrees of smutting this would probably give a higher loss than is actually suffered.

Etiology.—The loose smut of wheat is due to *Ustilago tritici* (Pers.) Jens., a systemic fungus, which infects the young ovary at the time of flowering and develops its mycelium within the seed, giving an intraseminal infection. The time of maturing and the dusty character of the smut heads are admirably adapted to the blossom infection. These smut spores are being blown about by the wind just at the time when the normal heads of adjacent plants are in the flowering stage. Many of the innumerable smut spores will fall upon the ground or upon various portions of the wheat plants, but some of the total number will lodge

between the glumes or chaff and reach the feathery stigmas, where they germinate in much the same way that pollen grains germinate and send an infection thread down the tissue of the style into the ovule, where it continues to grow within the young embryo plant, and becomes dormant when the seed is matured. Seeds carrying an intraseminal mycelium appear perfectly normal, but when planted the next season the dormant mycelium resumes activity with the awakening of the young plant, and keeps pace with the growth of the young seedling. According to Klush-

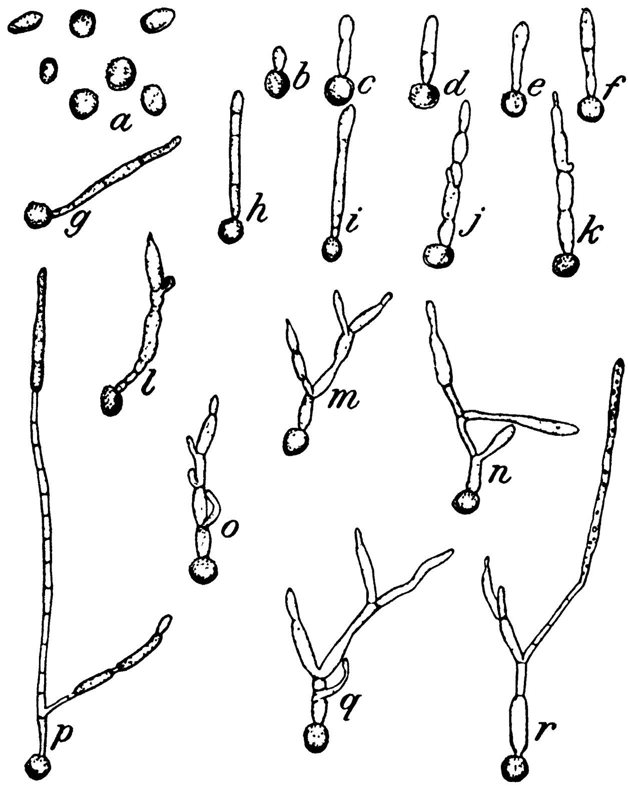

FIG. 211.—Spores of *Ustilago tritici* at rest and after various stages of germination in water. *a*, group of spores showing various shapes and sizes; *b–r*, early and later stages of germination. (*After Stakman, Minn. Agr. Exp. Sta. Bul.* **133**.)

kinova (1928), the mycelium is in all organs of the embryo, chiefly the growing point, and also the endosperm adjoining the scutellum; also in roots of seedlings up to the fourteenth to sixteenth day and in stems, leaves and sheaths in diminishing amount from the coleoptile to the apical leaves. With the organization of the heads, the mycelium makes a vigorous development, destroys the various parts, as noted under Symptoms, and gives rise to the dusty smut masses, or sori, consisting of globular or oval, pale, olive-brown, single-celled spores, 5 to 9μ in their

greatest diameter. These spores are generally lighter on one side and the wall, especially on the lighter side, is marked with minute spines.

Previous to the work of Maddox (1897) and of Brefeld and Falck (1905) it was supposed that seedling infection occurred in much the same way as has been described for stinking smut. The latter showed that spores applied to the seed or introduced into the soil were of no consequence in the production of the disease, but only spores applied to the blossoms were effective. This was demonstrated in two ways: (1) by introducing the smut spores into separate blossoms by means of a fine brush; and (2) by blowing the smut spores into cylinders in which blossoming heads were enclosed. Freeman and Johnson (1909) showed that

> Artificial inoculations of flowers with loose smut from the time when the stamens are still green to the time when the ovary is one-third its mature size are usually successful. The optimum period for artificial inoculation is the time when the flower is in full bloom or when the ovary is just commencing to develop after fertilization.

The spores of loose smut, as might be expected from their relation to infection, are not long lived as in bunt or other seed-borne or soil-contaminating smuts and germinate with the direct production of an infection thread, rather than by the formation of a promycelium, and secondary spores or sporidia. The spores are able to germinate freely in water or nutrient solutions as soon as mature, but are not able to retain their viability for more than 5 or 6 months, even suffering a very marked loss of germinative power during this period. It was first noted that the infection hyphæ are pushed out from the clear side of the spore, but Lang (1910) has noted two to three clear spots in the wall through which the infection hyphæ emerge. The spores of loose smut of barley and of wheat are morphologically quite similar, but in *Ustilago nuda* the mycelium formed in cultures produces straight lateral branches at right angles to the main axis, while in *U. tritici* the lateral branches are curved and more variable in their insertion (Riehm, 1914).

The smut spores that lodge on the feathery stigma behave much the same as pollen grains and

> . . . send their germ tubes into the latter only when its ephemeral cells have begun to collapse and dry up. The germ tubes then enter in and between the cells and pass down to the lower, still living, part of the style. Here many are checked but others continue through the intercellular spaces and the channels left by pollen tubes, until in a week or so the cavity of the ovary is reached. The ovules are next penetrated through their integuments, entry taking place through small intercellular spaces. The integuments ordinarily become cutinized and impermeable about 10 days after the normal time of infection, so that successful penetration of the ovule usually occurs between the seventh and tenth day. The infection tube then passes into the space between the endosperm and the

nucellus, where it branches freely for the first time. In about 3 weeks branches have reached the lower end of the raphe, and they pass around the bottom of the endosperm to reach the scutellum and penetrate the embryo. Here some of them grow along the rudimentary vascular bundles to occupy the part between the apex and the root, it being now about 4 weeks after infection. A month later all the parts of the embryo, except the root, contain hyphæ and there is a very copious mycelium in the scutellum. The hyphæ are mostly 2.5 to 3μ in diameter, except in the scutellum where they are often somewhat swollen; growth is exclusively intercellular; there are no haustoria; and the host cells are not affected in the slightest degree by the presence of the parasite. In the ripe grain, the hyphæ are thick-walled, oily and irregularly swollen; though no haustoria are found, the cell walls are indented in places (Butler, 1918).

According to Lang (1910, 1913), the mycelium does not even penetrate the cells of the host when it begins the organization of spores in the parts of the inflorescence, but simply pushes them to one side. Riehm (1914), however, found the mycelium penetrating the cells in the abnormal lesions on the vegetative parts.

The time of seeding of spring wheat does not seem to have any appreciable effect on the per cent of smut which appears in the crop, but data as to how time of seeding affects reinfection are not available. Some tests indicate that the time of seeding in fall grain influences the development of the parasite, the lowest per cent of smutted heads appearing in the late seedings. This can be explained by supposing that the low temperatures of late fall which are sufficient to induce germination of the seed are not sufficiently high to start the growth of the intraseminal mycelium, with the result that the growing point "runs away from the pathogene" and the pathogene is never able to catch up with it even though favorable temperatures may later start it into activity.

Biological Strains.—Recent cultural studies have led to the conclusion (Rodenhiser, 1926) that *U. tritici* and *U. nuda* are not distinct species but physiologic forms within the same species. On the basis of cultural characters only as "color, topography, character of surface, presence or absence of aerial mycelium, total amount of radial growth and type of margin" three strains from wheat and six from barley have been recognized. In more recent work (Grevel, 1930) pathogenicity on a series of differential host varieties has been used instead of cultural characters. Nineteen collections from Germany were placed in three physiological strains, while one from Turkey yielded a fourth strain. Twenty-nine others, including two from America, were placed in the three German strains.

Varietal Resistance.—Field experience has shown considerable variation in the percentage of loose smut in different varieties, but in 1909 Freeman and Johnson stated that no immunity of marked practical value had been discovered. Appel (1915) stated that immunity in loose smuts is due to the closed flowers, which prevent the smut spores from reaching

the stigmas during the susceptible period, but Fromme (1921) has pointed out that this does not hold for wheat, as none of the varieties has closed flowers. This structural explanation does, however, hold in the case of some varieties of barley and the loose smut peculiar to that species. Fromme (1921) has presented a brief comparative study of the variation of varieties and has shown that Leap wheat (Leap Prolific) under Virginia conditions shows either an entire freedom from smut or only traces under the same conditions which give 3 to 5 per cent in such susceptible varieties as Stoner. The amount of smut shown by bearded varieties was, as a rule, considerably in excess of that shown by the beardless varieties, the average for 20 of the former being 12.7 against 4.6 heads per row for 16 of the latter. Harvest King, Fultz and Gold Coin, all beardless varieties, also showed an apparent resistance to loose smut about equal to Leap.

Varietal susceptibility was studied by Tiemann (1925) with the recognition of very susceptible, moderately susceptible and immune varieties, with Strubes' Silesian, Wohltmann's Green and Hungarian Theiss in the last group. In a more recent study (Tapke, 1929) 102 varieties were tested by artificial inoculations. Hybrid 128, Little Club and Jenkin Club wheats were very susceptible, while Pentad, a Durum wheat, was found resistant. The "vulgare" varieties varied from high susceptibility to apparent immunity. Hussar was very resistant, while Ridit was immune. It seems probable that these tests were made with single biological strains; hence the conclusions may not be generally applicable.

There is no correlation in the H-ion values of the cell sap of resistant or immune and susceptible varieties. It has been shown by Piekenbock (1927) that resistance to loose smut is recessive and inherited according to the Mendelian ratio.

Prevention or Control.—Since the fungus is carried in the seed in the form of a dormant mycelium, the seed disinfection methods that are effective for bunt, smuts of oats or the covered smut of barley cannot be effective. When blossom infection was first established Brefeld was of the opinion that no seed treatment could be effective, because of the difficulty of killing the internal mycelium without killing the embryo. Jensen devised the hot-water treatment and first showed its effectiveness for smuts due to seed-borne spores and later proposed the *modified hot-water treatment*, which is still being recommended with only minor changes. The hot-water method is rather cumbersome and has not been generally recommended for the treatment of the entire lot of seed for large farm operations, but rather for the handling of small lots to be used for planting seed plots.

The modified hot-water treatment may be carried out as follows: (1) Soak the wheat in water at 68 to 86°F. for 4 to 6 hours; (2) immerse in warming vat for 1 minute at a temperature 5 to 10° under that of the

treating vat; (3) immerse in treating vat for 10 minutes at temperature of 129.2°F. (54°C.) or within a range from 124 to 130°F.; (4) drain and dip at once into cold water or spread out in a thin layer to cool and dry. Plant as soon as the seed will run freely through the drill, or dry thoroughly and store for later use. The exact method of providing the hot water and handling the grain can be varied. The amount of wheat treated at once has been varied from ½ peck to 1 bushel in wire baskets or burlap containers to as much as 5 bushels in a heavy wire drum which is arranged to revolve in a tank of water heated by steam. In Germany a special machine has been used in which the hot water is forced through the wheat (Appel and Gassner, 1907). Whatever type of container is used, it should not be filled, but should allow plenty of space for the quick and complete penetration of the hot water. A temperature of 135°F. for 15 minutes will be endured by wheat without serious injury, but it would be better to hold within the range specified. The treatment for loose smut of barley is the same, except that the immersion should be for 15 minutes at 125.6°F. (52°C.) with an allowable range from 124 to 129°F.

The value of the presoaking, according to Appel and Riehm, is due to the fact that the absorption of water starts the intraseminal mycelium into activity and renders it more sensitive to heat than when it is in the dormant state. The temperature of the water during the presoak period and the duration of the soaking are of importance, as has been shown by Appel (1909) and others, as may be illustrated by the following examples:

Presoak temperature, degrees Centigrade....	1.5	9	18	30
Per cent of smut.....	4.6	3.1	1.1	0
Hours presoaked..	2	4	6	
Per cent of smut.....	2.7	1 1	0	

Too low temperatures or too short periods of soaking will give imperfect control.

The limited range of effective and safe temperatures makes it imperative that thoroughly reliable thermometers should be used and every effort should be made to bring all of the wheat to the treating temperature at once by agitation, and to check the action of the heat at the close of the treatment period by rapid cooling. It would seem that the use of the cold bath should give less germination injury than simply spreading the wheat out to dry as in some of the more recent practices (Tapke, 1924).

The hot-water treatment, like chemical disinfection, causes more or less seed injury, and in this case also the injury of machine-threshed grain is greater than in hand-threshed grain. The amount of seed-injury is variable, depending on variety, threshing injury, care in treating, etc., but about what may be expected from the standard treatment may be judged from tests given by Tapke (1924), in which 33 untreated machine-threshed

samples gave an average germination of 87.6 per cent, while the treated lots showed an average germination of only 52.7 per cent. By careful adjustment of presoak temperatures and time and temperature of the dip, control has been obtained, with not over 10 per cent reduction of germination (Neill, 1925).

The single-bath, hot-water or steam treatment has been studied (Tapke, 1926) as a means of lessening seed injury. For the hot water a machine with an endless belt used by canneries, with the addition of a rotary grain drier, was employed. Treatments for 110 minutes at 45°C. (118.4°F.) or 95 minutes at 49°C. (120.2°F.) gave best control, with increased yield and less injury than with the modified hot-water treatment. For the steam treatment an upright grain drier of 2 bushels capacity was used, in which a saturated recirculating atmosphere of 46 to 48°C. was maintained. Control without seed injury and no decrease in yields were obtained, with 46°C. for 1 to 4 hours, 47°C. for 1 to 2 hours and 48°C. for ½ hour. These two methods are suitable only for cooperative plants handling large quantities of seed.

The hot-water treatment has been shown to be effective in preventing loose smut, and some of the earlier tests reported marked increases in yields from its use (Kellerman, 1891). The effect on yield must be considered in deciding whether treatment pays. In tests conducted for three years by Tapke (1924) "wheat grown from untreated seed outyielded that grown from treated seed when the rate of seeding was 6 pecks per acre," even in lots producing as much as 10 per cent of smut in the controls. When allowance was made for seed injury by increasing the rate of seeding, treated seed gave the highest yields. From these results it would seem that treatment would not pay unless allowance is made for seed injury and swelling of the seed (if seeded moist) by proportional increase in the rate of seeding.

In Indiana and Virginia the objection of the farmers to the cumbersome and rather difficult hot-water treatment has been partially overcome by the establishment of community treating plants (Pipal, 1921), making use of creameries, canneries, mill or other establishments furnishing live steam, or by the construction of special rotary machines.

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COMMON SMUT OF CORN

Ustilago zeæ (Beckm.) Ung.

Of the three smuts which affect corn, one, the so-called common or boil smut, has nearly a worldwide distribution, and is easily recognized by the characteristic smut masses or tumors which appear on all the aerial parts: ears, tassels, stems, leaves and occasionally on the brace roots. Another species (*Sorosporium reilianum* (Kühn) McAlp.) causes the *head smut* of sorghums and corn, on the latter confining its attacks to ears and tassels, while a little-known form (*U. fischeri* Pass.), causing a *kernel smut*, is reported from Italy and the West Indies.

History and Geographic Distribution.—Corn smut was mentioned by Bonnet as early as 1754 and Du Hamel and other French botanists were known to have been familiar with it some years earlier. It was described by another French scientist, Aymen, in 1760 and studied in more detail by him and also by Tillet, whose writings were published in 1766. The earliest record of corn smut in America was by Schweinitz (1822), who was the first botanist to do systematic collecting of fungi in the United States. Corn is native to America, probably Mexico, and was sent to Europe as early as 1500. Corn smut has undoubtedly occurred in America for centuries, but was described from France earlier than from America, because attention was then being directed to botanical studies. There are three historical periods in the study of this disease: (1) from the time of the first writings (1754) to the publication of De Candolle's important treatise on "Physiologie Végétale" (1832), during which corn smut was believed to be a physiological disturbance or an œdema, due according to Tillet to "too great an abundance of sap which in rich land is carried towards certain portions of the plant"; (2) from De Candolle (1832) to the final publication of the researches of the German mycologist, Brefeld (1895), during which the parasitic nature of smut was recognized, but infection believed to be seed borne, as in smuts of wheat and oats; (3) from Brefeld to modern times, this period being ushered in by the brilliant researches of Brefeld, which demonstrated that the disease was not systemic and seed-borne, but that infections were purely local and could occur in any actively growing tissues above ground. During the middle or De Candollean period important contributions were made by both French and German botanists.

Mention may be made of the first extensive studies of corn smut in the United States by Hitchcock and Norton (1896) in Kansas, by Arthur (1900) in Indiana and by Clinton (1900) in Illinois. The De Candollean concept made its impress on these studies, as all were concerned at first with seed treatments for control and attempts to produce infection by seed-borne spores. All obtained negative results from seed disinfection, thereby supporting the earlier conclusions of Brefeld that infection is from wind-borne sporidia which reach various aerial parts of the corn plant. In the following years minor additions were made to our knowledge of the disease by various workers, and more recently Piemeisel (1917) has studied factors affecting the parasitism of the smut fungus, Melchers (1921 and 1925) has suggested the possibility of biological strains and has reported on the life history and ecologic phases of the disease. Jones, Holbert, Hays, Garber and others have made special studies in the breeding of corn for smut resistance. The definite recognition of physiological strains has been a more recent accomplishment (Christensen and Stakman, 1926; Eddins, 1929), while the heterothallism of the smut fungus has been established.

Corn smut is prevalent in both North and South America to a greater or less extent wherever corn is grown. The same holds true of some European countries, but according to Magnus it did not reach middle Germany until 1875. It is recorded by Butler as common and destructive in parts of India, and rare or absent in other sections. In 1910 McAlpine reported that the common corn smut of Australia was the head smut, and that *Ustilago zeæ* was not known to occur in that country.

Symptoms and Effects.—The disease appears on the various aerial parts of the corn plant as either small or large tumors, at first whitish due to a covering membrane, but later dark and then black from the development of the enclosed smut mass. These smut tumors or sori may vary in size from minute pustules on the leaves to others, on the stalks or ears, as large as a child's head. When they reach maturity the covering membrane dries and breaks, exposing the dry, powdery mass of spores. Tumors may appear at almost any place where meristematic tissue occurs, but they are common on or near the midribs of leaves, at the junction of the leaf sheath and blade or at the nodal buds on the stem. Individual flowers of the female inflorescence or ear, groups of flowers or the entire ear may be involved. Sometimes the ovaries remain rudimentary or aborted or each may be hypertrophied to form a tumor. The surrounding floral scales may be involved and expanded into flattened structures which cover the less conspicuous ovaries. In the tassel or male inflorescence the individual organs may be converted into smut tumors, which are frequently elongated, irregular, greatly enlarged structures. The impression frequently prevails that only ears and tassels are smutted, but this is due in part to the conspicuous character of the tumors on these structures, and in part to the fact that in light attacks the flowers seem to be more generally affected. In this connection an actual count of 1,741 smutted plants with the distribution of the sori (Arthur, 1900) will be of interest (see the table on p. 748).

From these figures it may be noted that a larger per cent of the tumors were produced on the vegetative structures than on the reproductive structures.

Apparently, based on the researches of Chifflot (1909) and Iltis (1911), Butler states that

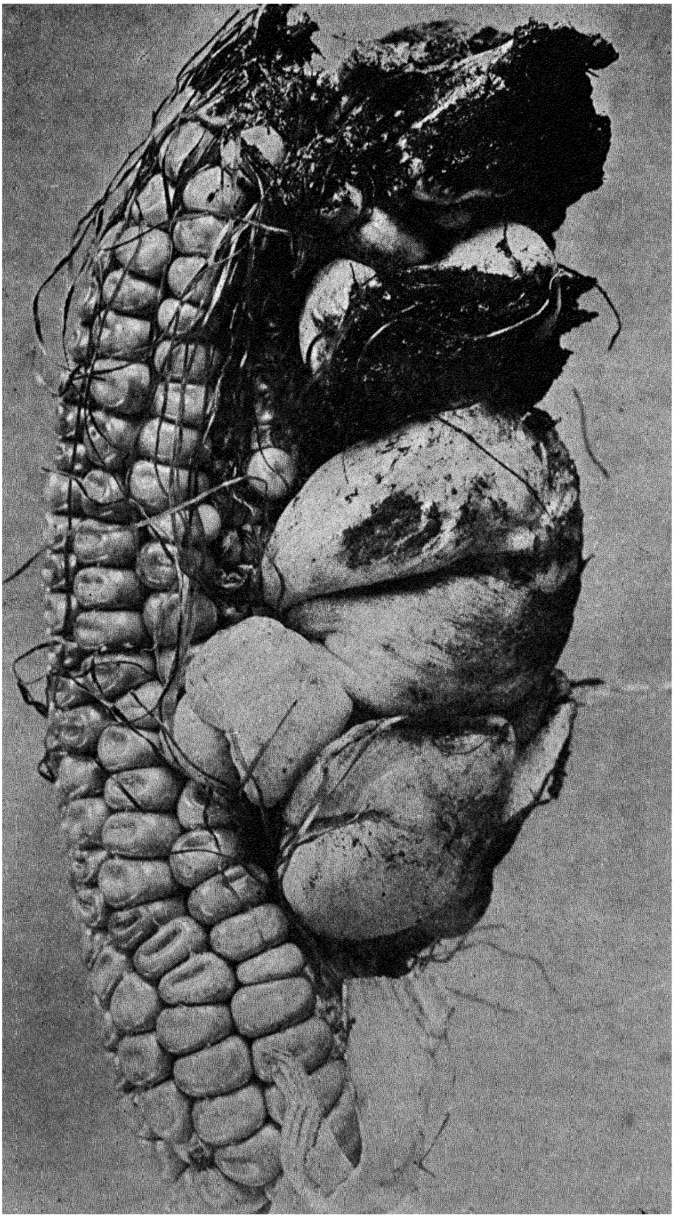

FIG. 212.—*Ustilago zeæ* on an ear of corn.

Deep-seated alterations may be caused in the inflorescence as a result of the attack. It is well known that in maize the male and female inflorescences are

separate, the former terminal, the latter lateral. In smutted plants, however, it has sometimes been observed that the male inflorescence bears female and hermaphrodite flowers in its lower part and may even give perfectly normal grain at the base.

Region of plant	Number of pustules	Percentage of pustules
Stem between first and fifth nodes[1]	753	37.2
Stem between fifth and tassel	342	16.9
Leaves (blade and sheath)	320	15.8
Ears	321	15.9
Tassel	287	14.2
Total number pustules on 1,741 plants	2,023	100.0

[1] According to Potter and Melchers these infections are not in reality on the stems, but on the buds at the nodes.

More recently, however, Werth (1913, 1919) has proved by experiments that the development of hermaphroditic flowers in the tassel is not related to the presence of smut.

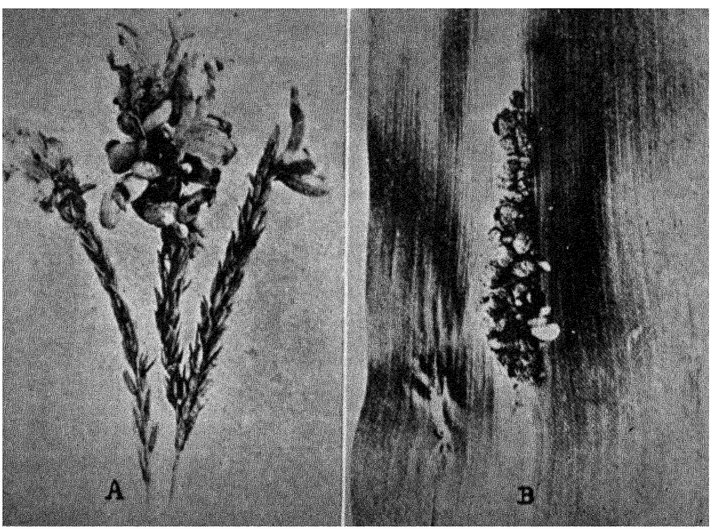

FIG. 213.—*Ustilago zeæ.* *A*, on corn tassel; *B*, on midrib of a leaf blade.

Loss from Corn Smut.—This is variable and rather difficult to measure. Corn smut may be present in traces only or a very high per cent of the ears may be smutted. The writer observed a field of sweet corn in Texas in which 60 per cent of the ears were smutted, and Bessey (1889) has recorded a loss of 66 per cent in sweet corn in Iowa. In both cases the land had been cropped to corn for a number of years. These figures

are high for field corn, especially if rotation is practiced. Losses of 5 to 15 per cent are recorded for Wisconsin in 1881, 20 per cent in parts of Hungary in 1902–1903, while Clinton (1900) estimated the loss for Illinois at ½ to 5 per cent. The Plant Disease Survey Records for the period from 1918–1924 estimate an average annual reduction in yield for the entire United States at 2.1 per cent (Melchers, 1925).

The injury manifests itself both directly and indirectly and will vary according to the number and location of infections. Large boils above the ear are more injurious than when located below. Smut infections on the stem reduced the yield of shelled corn from 7 to 54 per cent with a maximum of 94 per cent (Immer and Christensen, 1928). The greatest reduction in yield of plants of the same genotype was caused by induced sterility, one test showing 19.8 per cent sterility in smut-free plants and 38 per cent in smutted plants (Garber and Hoover, 1928). Smut also causes a pronounced reduction in sucrose and a lesser reduction of hexoses in the vegetative parts (Hurd-Karrer, 1927).

Etiology.—The common smut of corn is due to *Ustilago zeæ* (Beckm.) Ung., one of the true smuts, or Ustilaginales, in which the infections are localized, and not the result of a mycelium that has grown up through the tissues of the plant from the seedling stage as in the bunt or stinking smut of wheat. The fungus was named *Lycoperdon zeæ* by Beckmann in 1768, and this generic name will be recognized as that of some of our common puff balls. Other generic names were used by various early writers but Unger assigned the fungus to Ustilago in 1836.

A mature smut tumor consists of the thin covering membrane of host origin of thin-walled, uncuticularized, polygonal cells, with few distorted stomata and no trichomes. This encloses the mass of powdery spores mingled with remains of the old collapsed parenchyma cells of the host and fibrovascular bundles which make a fibrous network. The mature spore is spherical to ellipsoid, 7 to 12μ in diameter, with a brown wall covered with small spines. These spores are set free by the rupture of the enclosing membrane and may be mingled with the soil débris or scattered about by the wind. They may be carried to the feed lot with smutty corn fodder and there mingle with the barnyard manure. Either at once or after a period of dormancy the spores germinate if supplied with suitable moisture and temperature conditions. The immediate product of germination is not an infection thread, but a *promycelium*, which in its simplest form consists of four somewhat elongated cells from which both terminal and lateral *sporidia* are formed as somewhat fusiform bodies of variable size. Under favorable conditions of nourishment the promycelium may branch and a growth of some size may be developed, with an abundance of sporidia. Separated sporidia may germinate at once to form infection threads, or if supplied with a nutrient solution they will bud profusely in a yeast-like fashion, to form secondary

sporidia which may behave exactly like those produced directly from the promycelium. When promycelial filaments reach the air, sporidia are produced in great abundance in simple or branched chains, and as these are detached they are readily borne away by the wind. The original spores may be scattered by the wind and germinate wherever they find lodgment, and still further dissemination will result from the wind-borne sporidia. This extensive aerial dissemination of the sporidia was first demonstrated by Brefeld (1895). Whenever these sporidia reach young growing tissue of the corn plant they may germinate and produce localized tumors. Each tumor which is formed is the result of a separate infection, and the mycelium spreads but little from its original point of

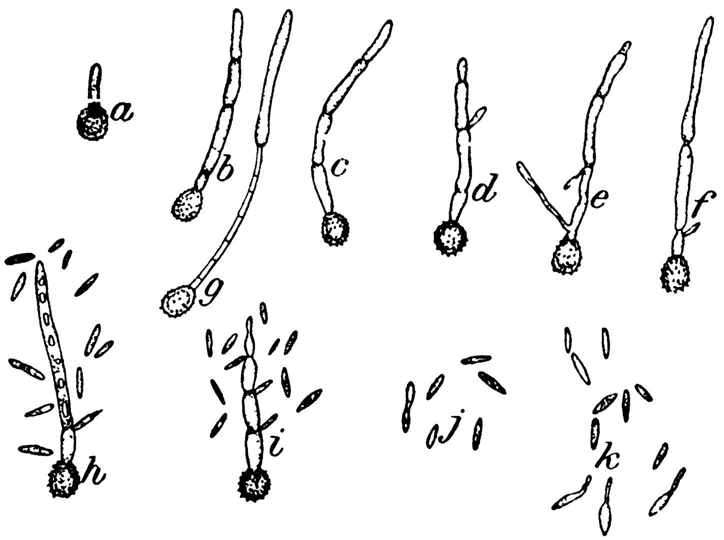

FIG. 214.—Germination of spores of *Ustilago zeæ*. *a–f*, germination in manure decoction at 19 hours; *g*, formation of germ tube in water at two days; *h* and *i*, promycelia at 7 days producing sporida; *j* and *k*, typical sporidia, the three lower in *k* germinating. *After Stakman, Minn. Agr. Exp. Sta. Bul.* **133**.)

entrance. The period between infection and the formation of mature sori varies from 7 days to 3 weeks, depending upon environmental conditions.

The germ-tube bores through the young epidermis and at first grows into and through the cells of the parenchyma below, stimulating them to active division. Some of the branches extend rapidly throughout the tissues, so as to spread the infection over a considerable area; these are known as *infection hyphæ*. Others form little clumps, with many branches, within individual cells; these are the feeding hyphæ, which may be compared to the haustoria of parasites that live mainly between the cells as the rusts of wheat. The infection hyphæ may either grow between the cells, sending feeding hyphæ into them, or may pass directly across a cell. In the latter case, the fungus does not, as a rule, come into contact with the cell contents, being enclosed in a cellulose tube or sheath which is

manufactured around the hyphæ the moment it enters the cell, and keeps pace with its growth. This sheath is developed through the activity of the cell protoplasm, and is a defensive device by means of which the cell endeavors to protect itself against the injurious action of the fungus. Sometimes it is formed so rapidly and in such strength as to stop the growth of the hypha altogether. In the young thin-walled parenchyma of the newly formed tumor, the bulk of the fungus is found chiefly between the cells, and up to the time of spore formation there is no destruction of the cell contents, the parasite living on surplus food supplied by the plant. The infection hyphæ, passing across the cells enclosed in their cellulose sheaths, are best seen towards the margin of the tumor. The hyphæ do not confine themselves to the cells of the parenchyma but also penetrate the young bundles, being found, for instance, in the large vessels of the axis of the inflorescence in diseased ears. They do not, however, extend far beyond the seat of primary infection, being limited by the swollen tissues of the tumor (Butler, 1918).

After the mycelium has permeated the tumor tissue, spore formation is initiated. The compact, branched hyphæ become segmented into short uninucleate cells, adjacent pairs becoming merged by the solution of the separating wall into irregular cylindrical, binucleate cells. In the meantime general gelatinization of the walls occurs and these binucleate masses of protoplasm surrounded by gelatinous sheaths constitute the spore origins. Each spore unit becomes rounded, surrounds itself with a spore wall, the gelatinous remains of the hyphæ are used up, the two nuclei unite and the isolated spores first constitute a pasty mass, which becomes powdery in the mature tumor.

It was formerly believed that the smut spores germinate only after passing through a period of winter rest, but numerous workers have shown that they will germinate if taken directly from new tumors in the middle of the summer. Piemeisel (1917) obtained an average germination of 42.8 per cent in tests made from June 24 to Oct. 10, but much higher in Cohn's nutrient solution (100 per cent or slightly less). Although they will germinate immediately, they are able to retain their viability for several years, and Brefeld records their germination when 7 years old, while Piemeisel obtained infection from material 5 years old. It is of importance to note that spores of corn smut lose their viability after having been kept in a silo for a few weeks, probably from the action of silage acids, especially acetic (Piemeisel, 1917). The relation of temperature to germination has been recently studied by Jones (1923). The optimum was found to lie between about 26 and 34°C., the maximum between 36 and 38° and the minimum was 8°. The optimum is higher than for some other smuts (see Bunt or Stinking Smut of Wheat) and probably explains why corn smut is more severe in warm corn-growing sections than in the cooler regions. Potter and Melchers (1925) have shown that corn smut is much worse in the dry, hot areas of Kansas than elsewhere.

It was formerly the belief that the spores could pass through the alimentary canal of animals without losing their viability. They are in large part or wholly killed according to Arthur and Stuart (1900), and the finding of these investigators has been substantiated by Ficke and Melchers (1929). The number of spores which remain viable after passing through the digestive canals of horses and cattle is so small that they may be considered negligible in the perpetuation and spread of the disease.

The sporidia have generally been considered as short-lived, and according to Arthur and Stuart (1900) "these are borne through the air, which must be rather moist or the sporidia will be killed by drying." Brefeld (1895) found that sporidia dried for 5 weeks were killed, but Piemeisel (1917) found that sporidia taken directly from pure cultures would withstand drying on cover glasses for 149 days at room temperature. If taken from a water suspension and dried, thus more nearly approaching natural conditions, they are more sensitive to desiccation. They still remained viable after being grown continuously in pure cultures for 3½ years. Freezing injures sporidia but little, as may be judged from tests in which they were exposed to −28°C. without losing their viability, but moist sporidia are killed by alternate freezing and thawing. They can germinate and bud in silage juice but are injured by solutions equal in acidity to silage.

Spores of corn smut will germinate in the compost heap and sporidia will continue to bud in water extracts of manure, in much the same way as in nutrient solutions. This budding of sporidia undoubtedly occurs to a greater or less extent under natural conditions in the barnyard or in the field, the exact behavior varying with the compositions of the solutions in which germination occurs. This behavior will explain how the enormous number of smut spores produced by a tumor may be increased and a more widespread dissemination brought about.

Under experimental conditions in the greenhouse young plants may be infected, either by hypodermic injections of sporidial suspensions or by dropping sporidial suspensions into apical buds (Tisdale and Johnston, 1926), and in many cases such infected plants are killed. Under natural field conditions infections do not take place until plants are 1 to 3 feet high, and new infections may appear any time up to the tasseling stage. This freedom of young plants in the field has been attributed to the temperatures being too low during the seedling period (Tisdale and Johnston, 1926), but this view is not held by Immer and Christensen (1928), who suggest that seedlings are either morphologically or physiologically resistant to field infection. During the susceptible period infection may take place at any point where there is young growing tissue, but matured tissues cannot be penetrated.

It has been shown that the corn-smut fungus is generally heterothallic; that is, monosporidial cultures are incapable of producing infection with the production of smut boils but may produce flecking. The use of plus and minus monosporidial cultures or those of opposite sex gives successful infection (Stakman and Christensen, 1927; Hanna, 1929). The presence of clamp connections in the parasitic mycelium within the host has also been observed (Seyfert, 1927), showing that the binucleate condition arises sometime before the formation of the chlamydospores.

Host Relations and Conditions Favoring Smut.—*Ustilago zeæ* affects but one other host besides common corn. It has been found under natural conditions on the related plant, teosinte (*Euchlæna mexicana*), and artificial infections have been successful. Differences in susceptibility of different varieties of corn have been noted. Pammel reported the great severity of smut on corn imported from Mexico, South America and the Philippines, and mentions the fact that it is supposed to be more severe in warm countries than in cold countries. In breeding work, Jones (1918) has described the differences in susceptibility of inbred strains of maize to smut attacks, and Hays *et al.* (1924) report an average of 1.9 per cent in two dent varieties and injury ranging from 3.9 to 13.4 per cent in three flint varieties. This is in keeping with previous statements that flint varieties are more susceptible than dent varieties. According to Melchers (1921), "It is being found that selections and crosses of corn showing resistance to smut in one locality or state are much more susceptible in other sections," and he attributed this largely to the physiological differences in the smut fungus, since some strains are known to be much more virulent than others.

Any conditions, such as rich, moist soil, which stimulate the host to vigorous growth with softness or increased succulence, increase the chances for infection. If conditions are favorable, the longer the growing season the greater the amount of smut. Therefore, early-planted corn generally smuts worse than a late-planted crop. Infection occurs very largely during either cloudy, moist days or during dewy nights. Showers followed by bright sunshine are less conducive to smut than moderate rains with slow clearing and moist nights. Close planting is favorable to smut, since the air remains moist for a longer time than in a thinner stand. More recent studies indicate, however, that the presence of moisture is not so important a factor for corn-smut infection as formerly believed. The importance of temperatures during the summer months has been pointed out (Platz, 1929), the season of 1927, with subnormal mean temperatures but very moderate rainfall, showing more smut than 1923 with higher temperatures and heavier rainfall. It has been demonstrated that ears with thin husks that split easily or are too short to cover the ear tip are more susceptible than ears with thick, long husks that completely enclose the ear (Kyle, 1929). Resistance to smut may be an

accompaniment of reduced vigor from whatever cause. "In selecting smut-resistant, selfed lines it must be borne in mind that smut resistance may be due to lack of vigor. The use of strains having such low vigor may result in lower yields" (Kyle, 1930). That resistance is in part physiological has been demonstrated by the inhibiting action of filtered juices of vegetative parts of resistant varieties upon the growth of the smut fungus in cultures (Ranker, 1930). Although smut can enter the unbroken tissue of the corn plant, infections may be greatly increased by mechanical injuries. MacMillan (1918) records a case in which smut was increased from 1 to 19 per cent by a hailstorm. The storm caused the increase by rupturing mature smut masses and scattering the spores and making numerous wounds through which infections resulted.

Physiological Strains and Mutation.—The first definite proof of the existence of physiological strains was presented by Christensen and Stakman (1926). Cultural studies from collections from various parts of the country yielded 15 forms that could be recognized by rate of growth, color, topography, surface, zonation, conidial production and margin. At least 7 could be recognized by their varying parasitic behavior on 10 selfed lines, the different strains showing great variation in their virulence. In cultures, sectors and patch mutants arise in most monosporidial lines, and these appear to be true mutants, often showing different morphological and physiological properties, including pathogenicity and sex properties. The isolation of 220 mutants within a year from one line, 162 of which were different, is offered as an illustration of frequency of mutation (Stakman *et al.*, 1929). The following conclusion is based on extensive studies: "*Ustilago zeæ* comprises an indefinite number of monosporidial or haploid lines that differ from each other in so many physiologic characters other than sex that they might well be considered physiologic forms."

Monosporidial lines from various foreign countries and the United States when paired with monosporidial lines of opposite sex from Minnesota produced smut galls (Stakman, Christensen *et al.*, 1929). They conclude: "The corn-smut pathogene, therefore, comprises many lines now, and new ones continually are arising by mutation and hybridization."

Corn Smut Not Poisonous to Cattle.—The belief has been and still is prevalent that corn smut is poisonous to cattle, and at one time it was looked upon with suspicion as having something to do with the so-called corn-stalk disease of cattle. Analyses of corn smut have shown that it contains more protein than corn, oat or clover hay, with a high content of carbohydrates (Smith, 1896). Feeding experiments have been carried out by workers at the Wisconsin, Illinois, Michigan and Kansas Experiment Stations and at the U. S. Department of Agriculture (Moore, 1896) and the results are summarized by Arthur (1900) as follows:

As eaten by animals in the field it is only rarely injurious; the action, when any occurs, affects the nervous system, and except in the unusual case of death causes no permanent injury. The alkaloid, or other active principle, occurs in corn smut in small amounts, and only under rare and exceptional circumstances is an animal likely to eat enough of the smut in any form to be affected by it.

Although corn smut has been reported to contain an active principle resembling that of ergot in its action, it is not present in sufficient amount to be of danger to cattle. Smith (1896) stated that feeding up to 11 pounds per day produced no signs of abortion in pregnant cows and that the milk yield remained normal.

Control.—Since it has been definitely shown that the smut does not result from seed-borne spores of the pathogene, seed disinfection can be of no value. This has repeatedly been proved by the early experimental tests. Since the infections are local and on aerial parts, spraying with a fungicide may be expected to give protection. It is interesting to note that experimental tests have shown that corn can be protected by spraying with Bordeaux. While a certain measure of protection by this treatment is possible, it has been considered too troublesome and too expensive to be introduced into farm practice and gives less protection when the host injury is taken into consideration than was formerly believed (Potter and Melchers, 1925). The remaining control practices for consideration are: (1) crop rotation or not planting corn more often than once in 3 years on the same land; (2) the collection and destruction of smutted ears or stalks before the spores have been disseminated; (3) the avoidance of smut-contaminated manure as a fertilizer for corn ground; (4) the adoption of planting and cultural practices which will be the least favorable to infection; (5) the use of resistant varieties. At the present time the main reliance is placed on crop rotation in the general corn belt. The destruction of the smutted ears and stalks may be practical and profitable in truck gardens, but would hardly be a paying operation for extensive field practice.

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IMPORTANT DISEASES DUE TO SMUT FUNGI

1. USTILAGINACEÆ

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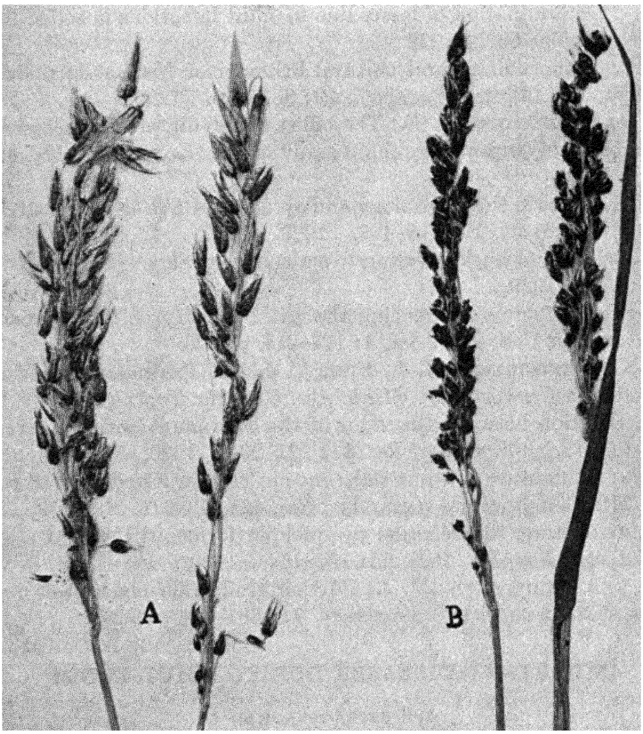

FIG. 215.—Oat smuts. *A*, kernel smut, (*Ustilago levis*); *B*, loose smut, (*Ustilago avenæ*).

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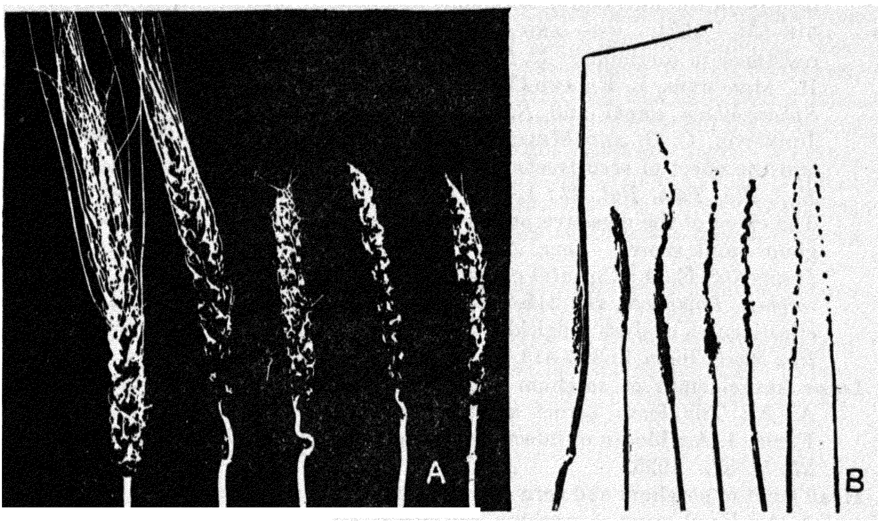

FIG. 216.—Barley smuts. ***A*, covered smut, (*Ustilago hordei*); *B*, loose smut, (*Ustilago nuda*).**

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2. TILLETIACEÆ

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Black smut of rice (*Tilletia horrida* Tak.).—ANDERSON, A. P.: A new Tilletia parasitic on *Oryza sativa* L. *Bot. Gaz.* **27**: 467–472. 1899. FULTON, H. R.: Diseases affecting rice. *La. Agr. Exp. Sta. Bul.* **105**: 24–28. 1908. BUTLER, E. J.: Fungi and Disease in Plants, pp. 226–228. 1918. TEODORO, N. G. AND BOGAYONG, J. R.: Rice diseases and their control. *Philippine Agr. Rev.* **19**: 237–241. 1926.

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White smut of spinach (*Entyloma ellisii* Halst.).—HALSTED, B. D.: Some fungous diseases of spinach. *N. J. Agr. Exp. Sta. Bul.* **70**: 1–15. 1890.

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CHAPTER XXV

DISEASES DUE TO RUST FUNGI

UREDINALES

The rust fungi or the Uredinales represent an order showing certain relationships with the true basidium fungi, and because of these affinities they have sometimes been designated as Protobasidiomycetes.

Nature and Importance.—The common name of the order has been suggested by the conspicuous orange or reddish spore pustules or sori which are characteristic of certain stages in the life cycle of typical forms. The true rust fungi are obligate parasites affecting a few ferns, but attacking in the main various species of seed plants (Gymnosperms and Angiosperms). The ravages of rust are known wherever plants are cultivated, species of the order being responsible for important diseases in nearly all groups of economic plants: cereals and grasses; forage crops; fruits of the garden and orchard; greenhouse and garden ornamentals; and trees of the forest. In addition to the species attacking economic plants there is a wealth of forms which are confined to weeds or to plants of no importance.

General Characters.—The following are the important characters of the group: (1) an intercellular, branched, septate mycelium (more rarely intracellular) containing yellowish or orange-red oil drops; (2) polymorphism of spores, typical rusts producing a succession of five different forms in the course of the life cycle; (3) the germination of the teliospore to form a *promycelium*, or a sporulating stage independent of the host, rather than the direct production of an infection hypha; and (4) the development in certain species of heterœcism, or the separation of the spore forms on two separate and unrelated hosts.

The Mycelium and Its Effects.—The internal, intercellular, septate mycelium is frequently brought into nutritive relations to the host cells by the formation of specialized sucking organs or *haustoria* which penetrate the cells. These haustoria may be globular, tubular, inflated, branched or in ball-like coils. Uninucleate and binucleate mycelial cells are characteristic of certain stages in the life cycle. The first effect of rust mycelium is not normally a killing of the host cells, but frequently a stimulating effect which may cause abnormal growth and continuation of vegetative development. When death of cells occurs it is generally delayed until the mycelium has developed spore fruits. The mycelium

may be purely local or it may spread extensively throughout special organs, certain shoots or even the entire plant. In the case of local development, it spreads radiately from a center of infection for a few weeks or a month and develops a spore fruit and then dies leaving a dead brown spot, or in other cases the mycelium may slowly advance after the first spore formation, and produce other spore fruits. In most cases when there is an extensive development of the mycelium from a single infection the host parts may be variously malformed, galls, hypertrophied stems or witches' brooms appearing with the suppression or deforming of leaves or flower parts. In many such cases the mycelium may be perennial within the host tissues and continue to advance as long as the host remains alive, or in other cases it soon pervades the entire plant and may continue to sporulate from season to season. In other cases the mycelium may require more than a single season to reach the sporulating stage, after which it dies.

The Spore Forms.—The following spore fruits and spores are produced in succession by a typical rust and each stage may be indicated by a symbol:

Symbol	Spore fruits	Spores	Stage
0	Pycnia (Spermogonia)	Pycniospores (Spermatia)	} Cluster cup
I	Æcia (Æcidia)	Æciospores (Æcidiospores)	
II	Uredinia (Uredosori)	Urediniospores (Uredospores)	Red rust
III	Telia (Teleutosori)	Teliospores (Teleutospores)	Black rust
IV	Basidia (Promycelia)	Basidiospores (Sporidia)	

As soon as mature or after a period of rest each cell of the teliospore under favorable conditions may form a four-celled filamentous structure, *the promycelium*, each cell of which gives rise to a *sporidium* or the fifth spore form. The names of the spore fruits and spores in parentheses were the ones generally used up until 1905, when Arthur proposed the other terms, which have now been generally accepted by American workers.

A rust may develop all types of spores (0, I, II, III, and IV sporidia) in the course of its life cycle, or some species may omit one or more forms. The kind of spore forms for a given species is generally constant and may form a basis of classification.

The *pycnia* are minute flask-shaped or disk-like receptacles and open by a narrow pore or ostiole, or in some cases by a wide pore. They are either subcuticular or subepidermal, and the cavity is lined by hyphæ from which rounded, oval or elongated, spore-like bodies, the *pycniospores*, are abstricted. These pycniospores are produced in large quantities and are extruded from the ostiole with a sweetish secretion in which they are embedded. The pycniospores were supposed to be male cells, hence the older names of spermatia and spermogonia.

The pycnia never appear alone but are always accompanied or closely followed by æcia or some other spore form.

The *æcia* are globular, cup-shaped, tubular or irregular fungous fruits which burst through the epidermis or periderm of the host. In the typical form an æcium consists of a membrane of fungous cells, the *peridium*, enclosing or surrounding the central fertile portion, or sporiferous hyphæ which produce the one-celled *æciospores* in chains. The æciospores are mostly with an orange content, polygonal in shape from

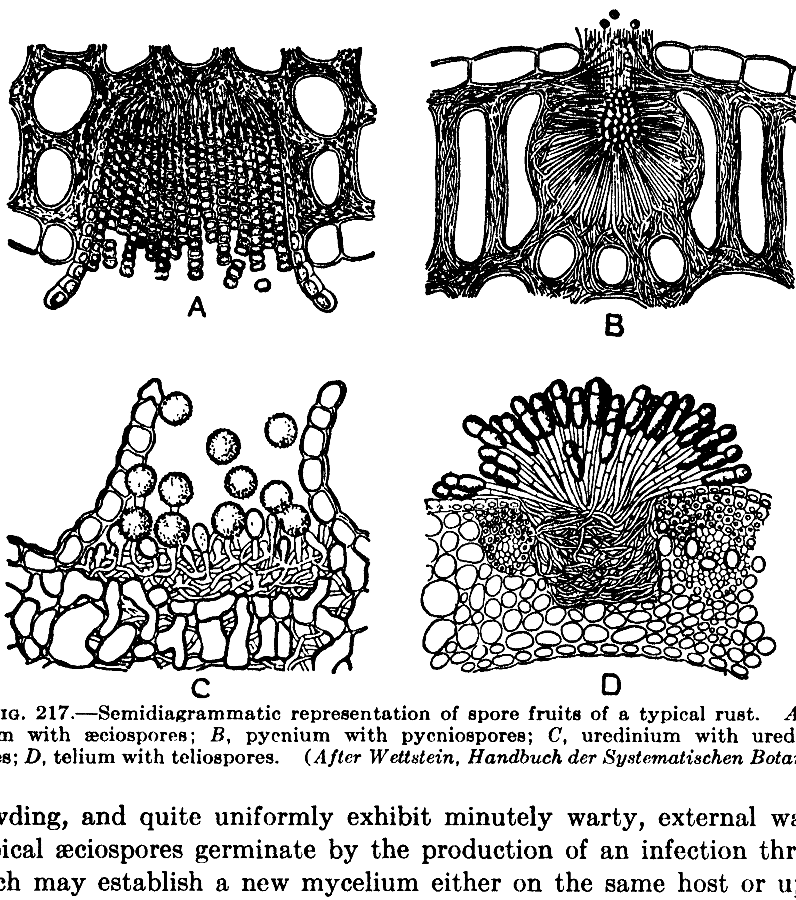

FIG. 217.—Semidiagrammatic representation of spore fruits of a typical rust. *A*, an æcium with æciospores; *B*, pycnium with pycniospores; *C*, uredinium with urediniospores; *D*, telium with teliospores. (*After Wettstein, Handbuch der Systematischen Botanik.*)

crowding, and quite uniformly exhibit minutely warty, external walls. Typical æciospores germinate by the production of an infection thread which may establish a new mycelium either on the same host or upon another one. This mycelium gives rise to either uredinia or telia, or in a few cases to secondary æcia. Four general types of æcia are recognized, based on the presence or absence of a peridium and its character: (1) peridium entirely absent or represented by a surrounding circle of sterile hyphæ or paraphyses (Cæoma); (2) body cup-shaped with the free or protruding edge of the peridium toothed (Æcidium); (3) peridium elongated-cylindrical, straight or curved, and split into segments which recurve in more or less star fashion when dry (Rœstelia); and (4) oblong,

globular or inflated with the peridium irregularly split or broken (Peridermium) and confined to the leaves and stems of Coniferæ.

The *uredinia* are groups of spore-bearing hyphæ crowded together to form spore pustules or sori, which become exposed by the rupture of the cuticle or the epidermis beneath which they are developed. Each short upright hypha or pedicel cell produces a single elliptical, ovoidal or spheroidal spore, a *urediniospore.* These spores are always unicellular, binucleate, with relatively thin, colorless or slightly brownish wall, marked on the surface with minute spines, needles or warts, and provided with one to 10 equatorial germ pores, while the contents are generally yellow or orange from the abundance of yellow oily material. Clavate or capitate sterile hyphæ or paraphyses are found mingled with the fertile cells in certain species. The urediniospores are easily detached from their pedicels and are able to germinate at once, forming infection hyphæ which enter the host through stomata. The resulting mycelium produces new uredinia, hence the urediniospores may be considered as a device for the rapid propagation of the species. They are formed in great profusion during the growing season and may be called the summer or repeating spores. They constitute the red-rust stage which is so conspicuous in the cereal rusts.

In certain species of rusts the urediniospores may show a changed structure and behavior. They may be thick-walled, have a persistent pedicel and germinate after a period of rest. These modified forms, which are called *amphispores,* are found in semiarid regions and are apparently devices for carrying the fungus through periods of extreme drought.

The *telia* are spore pustules or sori somewhat similar to the uredinia in general characters. They may be formed from the same mycelium or uredinia may be changed into telia. Telia generally follow uredinia when these appear in the life cycle, but they never appear earlier. The normal sequence of spore fruits frequently postpones the organization of telia well into the growing period, or in annuals towards the time of host maturity. Either sessile or stalked spores, the *teliospores,* are developed below the epidermis, in the epidermal cells or under the cuticle, and form sori of various forms and size, which may rupture the host tissue and expose the spores or remain embedded or covered. Telia are dark brown or black, powdery or gelatinous or form either cushion-like agglutinated layers or elongated columns. The teliospores are one to several or many celled, with relatively thick, dark walls that are smooth or variously sculptured with striæ, warts, reticulations, pits or spines or even extended into finger-like processes. Each cell of a compound spore is provided with one to four germ pores (one in the majority of species), is binucleate when young, but uninucleate when mature, and can germinate independently. When separated from the sorus the pedicels generally remain attached to

the basal cell. Germination of teliospores may occur in some species in the fall or as soon as mature, but in the majority of cases they must pass through the period of winter rest or be exposed to low temperatures before they can germinate. As contrasted to the urediniospores, which are repeating spores, for spreading the rust during the vegetative season, the teliospores serve the purpose of carrying the fungus over the winter period.

Species which normally form two-celled teliospores (Puccinia type) may produce many spores which are one-celled or similar to the Uromyces type. Such one-celled teliospores are called *mesospores*.

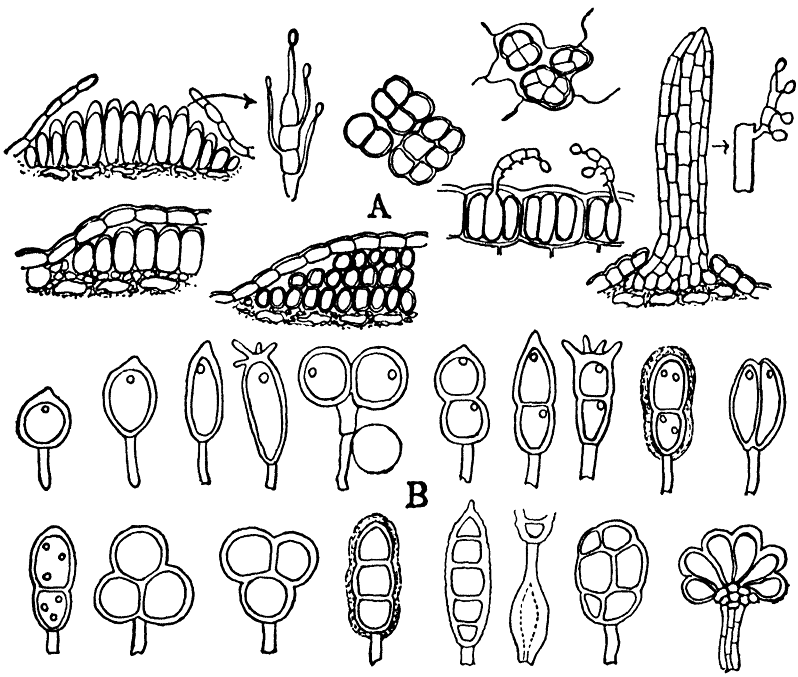

FIG. 218.—Types of teliospores. *A*, sessile or non-pedicellate forms; *B*, stalked or pedicellate forms. (*Adapted from various sources.*)

A single-celled teliospore or each cell of a compound spore may germinate by the production of a short or elongated hypha-like structure, a *promycelium*, which divides into four cells, from each of which arises a small thin-walled, ovate, globular or kidney-shaped spore, or *sporidium*, borne at the tip of a slender distal outgrowth or sterigma. A promycelium is sometimes considered as a *basidium* and the sporidia as *basidiospores*. The sporidia are forcibly detached from their sterigmata and are adapted for wind dissemination. In many cases they are relatively short lived. In autœcious species they may infect the same host, but in heterœcious forms they must be carried to the other or alternate host to produce an infection.

Heterœcism.—It is generally agreed that the occurrence of all the spore forms on a single host (the *autœcious* types) is a primitive character and that later the spore stages became separated in some way so that pycnia and æcia were developed on one host and uredinia and telia on another. This separation of the spore stages on two distinct hosts, generally entirely unrelated, is known as *heterœcism,* and finds a more complete expression in the rusts than in any other plants, although it is not uncommon in certain lower forms of animal life. It is estimated that over one-twelfth of all the species of rusts are heterœcious and new phases of heterœcism or new cases of heterœcism are frequently reported, based on either observations or experimental inoculations. Under natural conditions the two hosts are members of the same plant society, while in certain cases agricultural or horticultural practices have created serious disease problems by bringing two suitable hosts into close proximity to each other. In grasses, sedges, rushes and allied plants bearing uredinia and telia, the pycnia and the æcia will generally be found on some dicotyledonous host, while many dicotyledonous hosts bearing uredinia and telia may have their æcial stage on certain Coniferæ, although

Common name of the disease and scientific name of the rust	0 I Host	II III Host
Orange rust of aster and golden rod (*Coleosporium solidaginis* (Schw.) Thüm.)	Pinus spp.	Aster and Solidago species and a few other Carduaceæ
Blister rust of five-needle pines (*Cronartium ribicola* F. de Waldh.)	*Pinus strobus, P. monticola* and other five-needle pines.	Cultivated and wild species of Ribes (currants and gooseberries)
Poplar rust (*Melampsora medusæ* Thüm.)	Larix spp. (Larch)	Populus spp.
Witches' broom of fir and spruce (*Melampsorella elatina* (A. & S.) Arth.)	Abies and Picea species	Various species of Caryophyllaceæ or pink family
Rust of apple (*Gymnosporangium juniperi-virginianæ* Schw.)	Wild crabs and cultivated varieties of apples	*Juniperus virginiana* (red cedar); also on *J. barbadensis.* (III only) causing "cedar apples"
Pea rust (*Uromyces pisi* Wint.)	*Euphorbia cyparissias* (Spurge)	*Pisum sativum* (Pea) and *Lathyrus pratensis*
Rust of stone fruits (*Tranzschelia punctata* (Pers.) Arth.)	Hepatica, Thalictrum and Anemone species	Almond, apricot, cherry, peach and plum
Corn rust (*Puccinia sorghi* Schw.)	Oxalis	*Zea mays* (corn)
Rust of beet and spinach (*Puccinia sarcobati = P. subnitens Diet.*	Beet, spinach and 90 or more wild forms belonging to 22 families	*Distichlis spicata* (salt grass)

the disposition of the stages may be reversed. A few of the important cases of heterœcism among plants of economic importance may be tabulated.[1]

Life-cycle Combinations of Spore Forms.—The rusts may be divided into two groups depending upon the presence or absence of certain spore forms: (1) the short-cycle species; and (2) the long-cycle species. In the former the mycelium from a sporidium (basidiospore) produces pycnia which are followed at once by telia, or the pycnia may even be omitted. In the latter or long-cycle forms either æcia or uredinia or both are introduced between the pycnia and telia. The short-cycle types appear to represent a primitive condition, while the long-cycle types represent the highest specialization. The grouping of the spore stages and the terminology of the spore combinations may be presented in the accompanying tabulation.

Eu-forms		Opsis-forms		Brachy-forms	Hemi-forms	Lepto- and micro-forms	
Auto	Hetero	Auto	Hetero	Auto	Auto	Auto	Auto
0	0	0	0	0		0	
I	I	I	I				
II	II			II	II		
III	III	III	III	III	III	III	III
IV	IV	IV	IV	IV	IV	IV	IV
Long-cycle forms						Short-cycle forms	

Classification.—The Uredinales form a group of very closely allied species apparently of common origin. Two series may be recognized: (1) those in which the teliospores are sessile or without stalk (Impedicellatæ); and (2) those in which the teliospores are distinctly stalked or pedicellate, although the pedicel may be short or caducous (Pedicellatæ). There seems to be no uniformity of opinion as to the families which should be recognized, but grouping them in the following five families is convenient and affords a partial expression of relationships:

1. Endophyllaceæ.—Pycnia and æcia, with cup-like peridium or with peridium poorly developed or absent. Spores are produced from a fusion cell in chains with intercalary cells and are without perceptible germ pores as in typical æciospores, but germinate as soon as mature by a typical promycelium and sporidia instead of forming an infection hypha direct. Because of this behavior the spores may be called æcio-telio-

[1] See preceding page and also the special tabulation of the rusts of small grains, p. 794.

spores. This family includes the primitive rusts, the next step in advance being the separation of the single spore form into two: (1) the æciospores germinating conidially; and (2) the teliospores germinating basidially or by the formation of a typical promycelium. Two autœcious genera may be noted:

Endophyllum.—Peridium present, but sometimes poorly developed.

Kunkelia.—Peridium lacking, the æcio-telia of the Cæoma type.

2. Coleosporiaceæ.—Life cycle generally includes 0, I, II, III, and IV. Telia forming waxy, bright-colored crusts or cushions of sessile, unicellular teliospores seated on dilated hyphal cells. Teliospores of the indehiscent telia germinate in situ by the formation of three cross-walls in the somewhat elongated spore, while each of the four cells forms a long sterigma on which a large sporidium is borne. The four cells formed within the spore are the morphological equivalent of the typical external promycelium of other families. Urediniospores generally in chains. Two genera of the family may be noted:

Coleosporium.—Life cycle, 0, I, II, III and IV. Æcial stage on needles of two-leaved pines. II and III mostly on dicotyledonous hosts.

Gallowaya.—Life cycle III only, on Pinus.

3. Cronartiaceæ.—Teliospores unicellular, not pedicellate, produced in chains which remain separate or are united into lens-shaped, wart-like or columnar telia (Telial columns). Teliospores germinate with production of typical external promycelia. Uredinia naked or with a peridium. Urediniospores in chains or single. Two genera may be briefly characterized:

Chrysomyxa.—Telia pulvinate; urediniospores borne in chains.

Cronartium.—Telia forming cylindrical columns, horny when dry; urediniospores produced singly on pedicels.

4. Melampsoraceæ.—Teliospores seated on inflated hyphal cells but unicellular or divided by longitudinal walls into several cells (two to four), not pedicellate, produced singly (Urediniopsis) or forming extensive palisade-like layers one to several cells thick, which may be subepidermal, subcuticular or intraepidermal. Germination as in Cronartiaceæ. Urediniospores mostly single, rarely in short chains, frequently mingled with capitate or inflated spore-like paraphyses. Mostly heterœcious. Only the more important of the genera will be listed in the following key:

A. Teliospores unicellular, in a single layer:
  Life cycle with only telia . . . . *Necium*
  Life cycle with all spore forms:
    Teliospore walls colored . . . . *Melampsora*
    Teliospore walls colorless . . . . *Melampsorella*
B. Teliospores unicellular in more than one layer . . . . *Phakospora*
C. Teliospores two to four celled by longitudinal walls:
    Life cycle with all spore forms; teliospores subepidermal . . . . *Pucciniastrum*
    Life cycle with 0, I and III; teliospores intraepidermal . . . . *Calyptospora*

5. Pucciniaceæ.—Telia erumpent or covered, teliospores one to several-celled, borne singly on a pedicel or in groups on either simple or compound pedicels, free or embedded in a gelatinous matrix and dark colored or nearly colorless. Germination by the formation of typical promycelia. Urediniospores always solitary. Æcia with or without peridium. The family shows all degrees of polymorphism, various degrees of heterœcism and the most complete physiological specialization or differentiation of biological species that is known among fungi. Some of the genera containing important pathogenes will be presented in the following key:

A. Teliospores unicellular:
  Uredinia and telia subepidermal .... *Uromyces*
  Uredinia and telia formed by fascicles of hyphæ which protrude through stomata .... *Hemileia*
B. Teliospores two-celled by transverse partition:
  Teliospores free, two or more attached by fragile pedicels to a common stalk .... *Tranzschelia*
  Teliospores embedded in a gelatinous matrix .... *Gymnosporangium*
  Teliospores free, borne singly on long or short pecidels:
    Æcia of the Cæoma type .... *Gymnoconia*
    Æcia with distinct peridium .... *Puccinia*
C. Teliospores more than two-celled by transverse septa:
  Teliospores embedded in a gelatinous matrix .... *Gymnosporangium*
  Teliospores free:
    Wall faintly colored or colorless .... *Kuehneola*
    Wall dark, and usually coarsely verrucose:
      Life cycle with all spore forms .... *Phragmidium*
      Life cycle with 0, I and III, pedicel of teliospores not swollen .... *Earlea*

In the North American flora, Arthur recognizes three families of rusts: (1) Coleosporiaceæ; (2) Uredinaceæ, including Melampsoraceæ, Cronartiaceæ and Endophyllaceæ; and (3) Æcidiaceæ, synonymous with the Pucciniaceæ. The composite genera, Uromyces and Puccinia, have been split up into several genera on the basis of the spore forms in the life cycle.

Uromyces:
  Life cycle with all spore forms .... *Nigredo*
  Life cycle with 0, I and III .... *Pucciniola*
  Life cycle with 0, II and III .... *Klebahnia*
  Life cycle with 0 and III .... *Teleutospora*
Puccinia:
  Life cycle with all spore forms .... *Dicæoma*
  Life cycle with 0, I and III .... *Allodus*
  Life cycle with 0, II and III .... *Bullaria*
  Life cycle with 0 and III .... *Micropuccinia*

6. Uredinales Imperfecti.—Certain form genera are recognized when the telial stage is unknown. With the proof of the telial connection, the species assigned to the form genera are referred to their proper genera. The form genera are as follows:

Pycnia and æcia or æcia only:
  Peridium absent .... *Cæoma*
  Peridium present:
    Irregularly split .... *Peridermium*
    Body cup-shaped, margin toothed .... *Æcidium*
    Body tubular, margin fimbriate .... *Ræstelia*
Uredinia only or also pycnia and æcia .... *Uredo*

Biological or Physiological Specialization.—The development of physiological strains within certain morphological species is even more pronounced in the rusts than in the powdery mildews. Rusts may be monivorous, that is, they may be confined to a single host species or even variety, while many are plurivorous, that is, capable of infecting many different hosts which may be related or unrelated. Plurivorous species may be autœcious, as in *Puccinia malvacearum*, or in heterœcious species the æcia may be developed on several hosts and the uredinia and telia on a single host. The most extreme type of this class is illustrated by *P. sarcobati* (*P. subnitens*) with 90 recorded æcial hosts distributed in 22 families with uredinial and telial stage confined to the salt grass, *Distichlis spicata*. In other plurivorous forms there may be several hosts for both æcial stages and uredinial or telial stages, or the æcial stage may be confined to one or two hosts while the uredinial and telial stages affect many different hosts which are generally closely related. The development of biological species may be expected in plurivorous forms. The most extreme case of biologic specialization is found in the stem rust of cereals (*P. graminis*) (see discussion of this disease). The existence of numerous biological strains has also been demonstrated in the other cereal rusts: crown rust of oats (*P. coronata*), stripe rust (*P. glumarum*), barley rust (*P. dispersa*), orange leaf rust of wheat (*P. triticina*) and other rusts of less economic importance.

Heterothallism.—The recent studies of Craige (1927, 1931) and others have shown that certain rusts at least are heterothallic, that is, that both plus and minus mycelia are produced by the germination of the sporidia. Monosporidial infections in such cases produce pycnia, but generally no æcia are formed. If, however, pycniospores from pycnia developed on a plus mycelium are mixed with the nectar from a minus pycnium, or *vice versa*, æcia soon appear. In such heterothallic species the pycniospores seem to supply the opposite sex element to mycelia from monosporidial infections, but the exact mechanism of this phenomenon has not been demonstrated. Jackson (1931) has suggested that heterothallism may be the primitive and universal condition in the present long-cycled species, with pycniospores playing the part indicated, while in short-cycled species or in those with reduced life cycle which are homothallic, the pycnia would be functionless and may be expected soon to disappear from the life cycle. The development or occurrence of homothallism would explain the failure of æcia to appear in the life cycle, while

the persistence of pycnia in certain species is only the lingering of a structure supplying the "sex" element in more primitive species.

Cytology of Rusts.—The æciospores of the rusts are binucleate and the mycelium produced from these spores is made up of binucleate cells. This binucleate condition continues in the uredinial mycelium and the urediniospores are also binucleate. The mycelium which produces teliospores is also binucleate and the young teliospore cells are binucleate, but shortly before maturity, or at least before the formation of the promycelium, the pairs of nuclei in the teliospore cells fuse, so that these cells become uninucleate. With the production of a promycelium the fusion nucleus divides and the daughter nuclei divide again. The four promycelial cells are thus uninucleate and their nuclei migrate into the sporidia during their formation. The mycelium formed by the germination of the sporidia consists of uninucleate cells and the pycniospores are uninucleate. The exact process, following infection by the sporidia, leading to the development of sporulating æcia is not entirely clear. Recent investigations (Allen, 1930; Andrus, 1931) suggest that certain hyphæ which grow up from the æcial primordium function as trichogynes or receptive female hyphæ, while the pycniospores function as spermatia. The final result is the organization and production of the chains of binucleate æciospores. The stage of the rust from the sporidia (basidiospores) to the production of the fusion cell, the uninucleate stage, is designated as the gametophytic generation, while the balance of the life cycle is the sporophytic generation. In a typical rust there is then an alternation of gametophytic and sporophytic generations in the life cycle.

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STEM RUST OF GRAIN

Puccinia graminis Pers. (*Dicæoma poculiforme* (Jacq.) Kuntze)

This widespread rust of cereals affects wheat, oats, barley and rye as well as numerous wild grasses and produces the condition known as "red rust" or "black rust." This misleading term of "black rust" is frequently used and frequently figures in the market quotations on wheat in the daily press. Stem rust is a more appropriate term, as this species of rust is more or less closely confined to the stem and leaf sheath, and the term will serve to distinguish it from the other cereal rusts, all of which also have a black spore stage.

In addition to stem rust, wheat is also attacked by orange leaf rust and yellow stripe rust; oats by leaf or crown rust; barley by dwarf leaf rust and yellow stripe rust; and rye by brown leaf rust and yellow stripe rust. A tabular presentation of the distinguishing characters of all the cereal rusts will be presented, following the treatment of stem rust.

History.—The "blasting and mildew" of biblical times was undoubtedly due to rust fungi, and Pliny and other early Roman writers were familiar with "mildew" and attributed it and other misfortunes to the gods or the stars. According to Eriksson and Henning (1896), the Romans believed rust a curse sent on them because of wicked acts, and held a festival—the Robigalia or Rubigalia—on Apr. 25, to propitiate Robigus or Rubigo, a special rust god, and thus protect their fields. As late as 1733 rust was not believed to be of fungoid nature, for Tull attributed it to the attacks of small insects "brought (some think) by the east wind," which feed on the wheat and leave their excreta as black spots on the straw. Stem rust was recognized as due to a fungus and first named *Puccinia graminis* by Persoon in 1797.

The stage on the barberry was then supposed to be an entirely distinct fungus and was named *Æcidium berberidis* Pers. Years before the genetic connection between these two forms was known, the barberry was supposed to exert an injurious influence on wheat. "Various suggestions were made as to the cause of this: some affirmed the barberry bush exhaled a noxious effluvium; others that the pollen of its flowers poisoned the wheat; others again that it appropriated to itself all the nourishment from the soil in its vicinity" (Plowright, 1889).

As early as 1805 Sir Joseph Banks wrote: "Is it not more than possible that the parasitic fungus of the barberry and that of wheat are one and the same species, and that the seed is transferred from the barberry to the corn?" So certain was the belief in the injurious effect of the barberry that laws were passed for its extermination or forbidding its planting. Such a law was enacted in Connecticut in 1726, and Massachusetts passed a barberry law in 1755, because "it has been found by experience that the blasting of wheat and other English grain is often occasioned by barberry bushes to the great loss and damage of the inhabitants of this province" (Mass. Province Laws, 1754–1755).

Marshall, working in England, determined by actual experiments that the barberry was able to cause rust in grain (1781–1784). This was done by planting barberry bushes in the middle of a field of wheat. The honor of being the first to demonstrate the connection between the barberry *Æcidium* and the *Puccinia* on wheat belongs to Schœler, a Danish schoolmaster. He made many experiments (1807–1817), but among others carried rusted barberry leaves enclosed in a box and rubbed their

under surfaces on rye leaves in the middle of a field when the leaves were wet with dew. In 5 days the inoculated plants were badly rusted, while there was no rust at any other places in the field. His results were published by the Royal Agricultural Society of Denmark (1818), but this paper was almost overlooked for many years.

At first uredinial and telial stages were also supposed to belong to distinct species of fungi, the former being described under the name of *Uredo frumenti* Sowerby. Tulasne (1854) demonstrated the genetic connection between the uredinial and telial stages in stem rust, and also showed that this relation was a general rule among Uredinales. It remained, however, for the classical researches of De Bary to demonstrate the heterœcism of stem rust and of various other species (1864–1865). He reproduced the Æcidium on the barberry by inoculating with black rust and then carried it back to rye by using the æciospores.

Following the botanical corroboration of farm experience as to the relation of the barberry to stem rust, laws were passed by Denmark (1869), Prussia (1880) and France (1888) which made possible the extermination of the barberry or restricted its planting, but in 1903 Denmark passed a more drastic law which stated that "Barberry bushes may only be grown in such botanical gardens as serve as part of an educational establishment" (Lind, 1915). The history of the barberry in its relation to stem rust in the United States falls into three periods: (1) an early colonial period, in which special laws were enacted requiring or making possible the extermination of barberry bushes (1760); (2) a period of indefinite beginning, in which these old laws were allowed to go without enforcement and no new eradication measures were adopted, presumably because of a growing belief that the barberry was only a minor factor in the production of rust epiphytotics (period ended with the adoption of the Barberry Eradication Campaign by the Federal Department as a special war measure); (3) from 1917 to the present, during which all 13 states in the eradication area (Ohio, Indiana, Illinois, Michigan, Wisconsin, Minnesota, Iowa, Nebraska, North Dakota, South Dakota, Colorado, Wyoming and Montana) have passed laws requiring barberry eradication, and are protected against the shipment of harmful barberries from outside territory into their confines by a special Federal quarantine, dated May 1, 1919. In cooperation with the states mentioned, the Office of Cereal Disease Investigations of the U. S. Department of Agriculture has been conducting a vigorous and systematic campaign within this eradication area for the extermination of all barberries harboring the stem rust. Antibarberry legislation was also passed in Alberta, Manitoba and Saskatchewan, the barberry being placed on the noxious-weed list.

Two other lines of study of stem rust have been prominent in recent years: (1) the recognition of specialized strains or races—biological or physiological species—begun with the work of Eriksson on *formæ speciales* (1894); and (2) testing and selection for rust resistance followed by breeding to obtain rust resistance. These specialized strains were called "Schwester-Arten" (Schroeter), biologische Arten (Rostrup, 1894) and physiological races (Hitchcock and Carlton, 1894). Eriksson in 1894 recognized five "formæ speciales" in Sweden. Carleton in 1899 distinguished three physiological races on wheat, oats, barley and rye: (1) *tritici* on wheat and barley; (2) *secalis* on rye; and (3) *avenæ* on oats. Freeman and Johnson added a fourth biologic species (1911), which has since been held invalid. Stakman and Levine (1922) separated a single one of these, *Puccinia graminis tritici*, into 37 biological forms by means of their action on 12 "differential hosts," *P. graminis avenæ* of oats into five biologic forms (1923) and *P. graminis secalis* into several. Since that date the number of biological forms has been greatly increased by the workers from Minnesota and other wheat areas: 100+ for *P. graminis tritici*, 7 for *P. graminis avenæ*, and 14 for *P. graminis secalis*.

Many observations have been made since the difference in the resistance of wheat varieties was first made in 1841, but it is only in recent years that systematic testing

of varieties for comparative resistance has been carried out, and selection and breeding experiments for rust resistance have been in progress in North Dakota (Bolley, 1909), in England (Biffen, 1907) and, since 1907, by Canadian workers and by the U. S. Department of Agriculture in cooperation with the Minnesota, Kansas and other experiment stations.

Geographic Distribution.—The stem rust of wheat is present in greater or less amount in practically every country in which wheat is grown. The records show that epiphytotics have been prevalent in England, Denmark, France, Germany and other European countries when barberries were commonly cultivated, but that they have ceased entirely or become infrequent in those countries since the systematic eradication of barberries has been practiced. In Australia and South Africa the stem rust has been prevalent and serious even though the æcial stage on the barberry is unknown or rare in that country. In the United States, "the stem rust of wheat is of great importance in the hard-winter- and hard-spring-wheat belts of the Great Plains area and in the states bordering the Ohio River. In Maryland, Virginia and other eastern states it has been almost entirely absent for many years, but is by no means unknown. In the interior mountain valleys, between the Rocky Mountains and the Sierra Nevada Mountains, and in the non-irrigated area of the Great Plains, it is only occasionally found and is seldom serious. In the interior valleys of California it is occasionally epidemic, though usually of slight importance. On the coast of California it is always present and almost always virulent. In the southern states only a small quantity of wheat is grown, and here the rust is often severe. In the southern half of Texas it makes wheat growing a hazardous undertaking, and even in northern Texas it is a factor of great importance. The greatest rust epidemic of the last decade, which was due to the stem rust of wheat, occurred in 1904 and extended over the entire Mississippi Valley and up into the wheat fields of the Canadian Northwest, being particularly severe in the spring-wheat belt. It invaded the dry lands west of the Rocky Mountains and was severe in the interior valleys of California" (Freeman and Johnson, 1911). In certain sections the æcial stage on the barberry is either rare or does not occur at all, as in Australia, central India, South Africa, the southern United States and the Inland Empire of the Pacific Northwest. In some of these localities stem rust often is severe, while in others—the Inland Empire of the Pacific Northwest, for example—the disease is only rarely of any economic importance.

Symptoms and Effects on Grains.—The onset of the disease in a typical case on a susceptible host is characterized by the appearance of elongated brown or reddish-brown, granular pustules which may burst through the epidermis of any portion of the plant, but are most abundant on the stem and leaf sheath, but not uncommon on the leaf blades and glumes. This localization of these pustules or sori on the stem has suggested the common name "stem rust," now generally in use. These pustules of the "red rust" or summer stage may appear without any surrounding chlorotic or dead cells, or in certain cases individual sori or groups of sori may be seated first in a chlorotic area which soon becomes dead, the surrounding tissue exhibiting a condition of hypersensitiveness to the presence of the rust. In many of these hypersensitive areas the sori remain small and poorly developed and frequently do not exhibit the characteristic elongated form. In the extreme cases of resistance, a rust infection may be marked by minute yellowish or brownish "flecks"

which never develop the typical red-rust stage, and their nature as rust infections can be detected only by microscopic examinations.

As the season advances, the reddish pustules are gradually replaced by black pustules or sori, which occupy the same position or burst through the epidermis adjacent to red-rust pustules. This is the winter or "black-rust" stage.

Rust sori may be few in number or they may be very numerous and, when numerous, adjacent sori may coalesce to form more or less elongated brown or black powdery streaks. The "red" spore powder is very easily detached, and the garments of anyone walking through severely rusted grain fields may be covered with the reddish brown-dust.

In harvesting badly rusted grain, the spores flying thickly in the air sometimes cause considerable irritation in the nostrils and throats of the men who are at work. This is, however, merely a mechanical irritation, occurring only when there is a great abundance of the rust (Carleton, 1905).

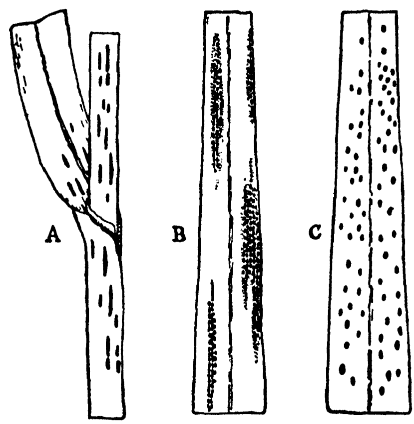

FIG. 219.—Diagrams showing form, size and arrangement of the uredinia of the three rusts of wheat. *A*, *Puccinia graminis*; *B*, *P. glumarum*; *C*, *P. triticina*.

The damage done by rust may be negligible in case of light or late attacks or almost a complete failure may result. The injury is due primarily to two disturbances in the nutrition of the host: (1) to the appropriation of food by the rust pathogene directly from the host cells in which it is living; and (2) to the increased loss of water, due to the rupture of the epidermis by the rust sori. The photosynthetic power of the host is impaired and the shortage of moisture results in poorly filled heads and shriveled grains. Rust infections in connection with other fungi have been shown to cause considerable sterility (Johnson, 1912). This means, then, a reduction in both the quantity and the quality of the threshed grain. This reduction in yield lags somewhat behind the actual percentage of rust; for example, 10 per cent rust gave 6.8 per cent reduction in yield (Goulden and Greaney, 1930). Where drought and hot weather coincide with a severe rust attack the rusted grain suffers from drought much more severely than normal grain, and burning, with its injurious effects, is the result. According to Weiss (1924): "Accompanying the reduction in yield, there is practically as great use of water as in healthy plants, that is, rusted plants have a higher water requirement, based on yield of both tops and grain." Weiss places more importance on the drain on elaborated food reserves as a cause of reduc-

tion in yield than on increased transpiration due to rupture of the epidermis by the rust sori.

Symptoms and Effects on the Barberry.—The barberry stage of stem rust first shows as a small, circular, yellowish spot upon the upper surface of the leaf, which increases in size to 2 to 5 millimeters or slightly more. The affected leaf tissue is hypertrophied, becomes margined with a brighter color or reddish purple and shows a central cluster of minute honey-colored pustules on the upper surface (later turning black), from which ooze minute droplets of pycnial nectar, and a group of minute cups with ragged or saw-toothed edges, the *æcia* or *cluster cups,* on the under surface. Similar cluster-cup lesions may appear on the fruits and fruit pedicels, and cause more or less hypertrophy of these structures. This stage generally makes its appearance in June or July, the exact time varying in different regions. The number of lesions per leaf may be only one or two, or they may be much more numerous. They are rarely sufficiently abundant to cause serious injury to the barberry, consequently the concern which this phase of the disease causes is due to the passage of the rust from the barberry to one of its grass or cereal hosts.

Economic Importance.—While rust has taken a regular annual toll in much of the wheat-producing country of the world, its greatest injury results in years of widespread epiphytotics. During the epiphytotic of 1904, much of the grain produced in the northern sections of the Mississippi Valley was so badly shriveled that it was entirely rejected by grain buyers, the yield dropping to as low as 4 bushels per acre (Carleton, 1905). In this epiphytotic the reduction in yield for Minnesota, North Dakota and South Dakota, the important portion of the spring-wheat belt, was estimated in excess of 23,000,000 bushels, valued at nearly $16,000,000.

Year	Estimated stem-rust losses to wheat	
	Bushels	Value
1915	14,000,000	$ 12,175,000
1916	184,208,000	283,697,000
1917	9,906,000	19,838,000
1918	665,000	1,341,000
1919	41,766,000	96,446,620
1920	51,973,000	67,850,020
1921	19,156,000	17,289,340
1922	18,868,000	17,578,210
1923	33,052,000	28,354,240
1924	5,856,000	7,484,720
Total	379,450,000	$552,154,150

Estimated losses from stem rust of wheat in the 13 barberry-eradication states (Colorado, Indiana, Illinois, Iowa, Michigan, Minnesota, Montana, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin and Wyoming) for the 10-year period from 1915 to 1924 as presented in the table on page 778 will serve to emphasize the magnitude of the rust problem during recent years.

The year 1916 was the outstanding rust year of the period covered, with a loss in the United States of over 180,000,000 bushels, while in Canada the loss for the same year amounted to about 100,000,000 bushels (from data compiled by Dr. E. C. Stakman). The average annual loss in the barberry-eradication states for the 6-year period 1915–1920 is estimated at more than 50,000,000 bushels, while for the next six-year period the estimated loss was reduced to about 15,000,000 bushels (Stakman *et. al.*, 1927). This may be credited in part to the eradication of barberries and in part to less favorable rust years. Losses have continued to drop up to the present.

Etiology.—The stem rust of cereals and various wild and cultivated grasses is caused by the heterœcious rust, *Puccinia graminis* Pers., or, according to Arthur's recent nomenclature, *Dicæoma poculiforme* (Jacq.) Kuntze, which produces its *pycnial* and *æcial* stages on the common barberry, *Berberis vulgaris*, and to a minor extent on some other species, and its uredinial and telial stages on wheat, oats, rye, barley and about 75 wild and cultivated grasses. Within the morphological species *P. graminis* the following biological species are recognized:

1. *P. graminis tritici* Eriks. & Henn. on wheat (Triticum species) and barley, rarely on rye; also on many wild grasses, Hordeum, Agropyron, Bromus and Elymus species. Thirty-seven physiological strains of *tritici* were recognized by Stakman and Levine (1922) on wheat (*Triticum vulgare, T. durum, T. compactum*), emmer (*T. dicoccum*) and einkorn (*T. monococcum*), but the number has since been increased to over 100 (Wallace, 1932).[1]

2. *P. graminis avenæ* Eriks. & Henn. on oats; also on a number of wild grasses. Seven physiological forms of *avenæ* were recognized by Stakman *et al.* by the use of three differential hosts (1923) but at least 8 are now known. This strain can infect barley and rye in artificial cultures, but only so rarely and weakly that it is probably of no importance on these hosts in nature.

3. *P. graminis secalis* Eriks. & Henn., on rye and barley and many wild grasses (14 biologic forms).

4. *P. graminis phleipratensis* (Eriks. & Henn.) S. & P. on timothy (two biologic forms) and a weak parasite on oats, rye and barley; also common on Festuca spp. in nature.

[1] *Phytopath.* **22**: 105–142. 1932.

5. *P. graminis agrostis* Eriks. on *Agrostis canina* and *A. stolonifera*.
6. *P. graminis poæ* on blue grasses, *Poa compressa* and *P. pratensis*.
7. *P. graminis airæ* on *Aira cæspitosa* and *A. bottnica*.

According to Stakman there is no evidence that *P. graminis hordei* F. & J. on barley, wheat and rye, as described by Freeman and Johnson (1911), is a valid biologic species.

The enormous number of physiological forms has arisen in part by mutation (Newton and Johnson, 1927; Stakman *et al.*, 1930) but probably to a much greater extent by hybridization (Waterhouse, 1929*a*; Newton *et al.*, 1930; Stakman *et al.*, 1930). Since hybrids are so readily produced, experimentally, it is only reasonable to expect that similar hybridization occurs in nature. It seems that physiological strains may flourish for a time in a given region, and then subside to have their places taken by new ones.

In the typical life cycle of stem rust the æciospores from the barberry, if carried by the wind to some susceptible cereal-grass host, germinate and reproduce an infection, *uredinia* soon appearing with the production of numerous *urediniospores*. These spores are scattered by the wind or other agents and spread the trouble, on the same host or on other susceptible hosts, and are thus repeating spores, being responsible for the extensive spread of the fungus during the growing season. The *telia* produced later in the season form *teliospores*, which remain dormant during the winter and in the spring germinate and produce typical promycelia and sporidia. The sporidia are forcibly abjointed and carried by the wind to the barberry, on which *pycnia* and *æcia* are formed.

The mycelium of the rust develops in the intercellular spaces and sends small rounded or branched *haustoria* or absorbing structures into the adjacent cells, and thus obtains its food directly from living protoplasm. The mycelium from a single infection has a limited range, but soon becomes massed beneath the epidermis and organizes a sorus. From this hyphal aggregate, short, erect branches arise which give rise to the urediniospores. With the further development, the covering epidermis is pushed up and is finally ruptured, exposing the urediniospores, which are soon set free. Typical mature uredinia are linear, 2 to 10 millimeters or more long, pulverulent, yellowish brown and surrounded by the cleft epidermis. The urediniospores are ovate, oblong to ellipsoid, echinulate, 14 to 22 by 17 to 45μ and generally have four equatorial germ pores.

Under favorable conditions of moisture and temperature the urediniospores lodged on a susceptible host germinate, each sending out two germ tubes, one of which usually makes a more vigorous growth than the other. With short duration of moisture, for example three hours, only very slight infection results, but if favorable moisture is maintained for 24 hours, the infection may reach 90 per cent, while over 24 hours'

duration of moist conditions may result in 92 to 100 per cent infection (Peltier, 1925). These germ tubes grow over the surface of the epidermis, and when a stoma is reached the tip swells up into an elongated vesicle, the *appressorium*, which lies over the slit-like mouth of the stoma. The contents of the germ tube become massed in the appressorium, which soon forms a fine branch that penetrates the stomatal opening and swells

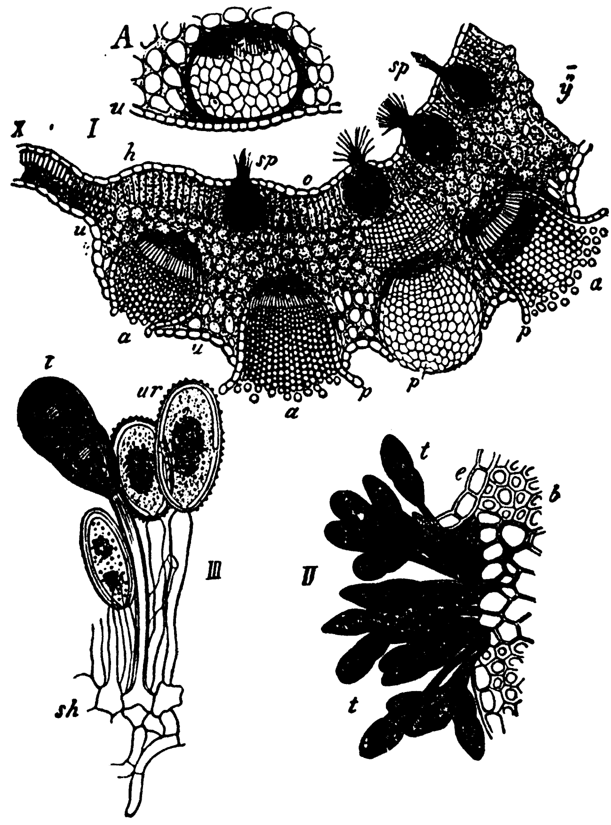

FIG. 220.—Spore fruits and spores of *Puccinia graminis*. I, cross-section of a barberry leaf showing æcia (*a*), pycnia (*sp*), peridia of the æcia (*p*), upper epidermis (*o*), lower epidermis (*u*), normal thickness of leaf (*x*) and thickness of hypertrophied portion (*y*); *A*, a young æcium; II, section of a telium, showing teliospores (*t*), epidermis of host (*e*) and subepidermal sclerenchyma fibers (*b*); III, portion of a uredinium with urediniospores (*ur*) and one teliospore (*t*). (*A and I after Sachs; II and III after De Bary.*)

up in the substomatal chamber to form a "substomatal vesicle." The contents of the appressorium and external hypha pass into the substomatal vesicle and this gives rise to one or more infection hyphæ which grow toward adjacent cells, and establish connection by haustoria. Infection is now completed and the mycelium spreads rapidly from cell to cell, but large leaf areas are not involved, the large numbers of sori representing numerous infections. Under optimum conditions for growth, uredinia with mature spores may be developed in 6 or 7 days.

The whole appearance of both fungus and host during the first few days after infection indicates a fairly perfect relation between the two. The fungus flourishes vigorously, while for a considerable length of time the host cells, even in the infected area, are apparently quite healthy. In no case does there appear to be an extensive killing of host tissue (Stakman, 1914).

The same mycelium which produced uredinia, may give rise to telia later in the season, the sori gradually changing to black as the urediniospores disappear and the teliospores take their place. The teliospores are chestnut brown, oblong-clavate, rounded or attenuate at the free end, which is much thickened (6 to 13μ), two-celled, slightly constricted at the cross-septum and firmly attached to the brownish persistent pedicel (up to 60μ long). They measure 35 to 65 by 11 to 22μ, and each cell is provided with a single germ pore, one at the apex of the terminal cell, the other just below the cross-partition. Teliospore formation is more rapid at 70 to 75°F. than at 55 to 60°F., but those formed at the latter temperature show an earlier and better germination (Johnson, 1931). The teliospores are not capable of germinating at once, but are resting spores, designed to carry the pathogene over to the following season. The period of dormancy can be greatly reduced by freezing and by alternate wetting and drying. The shortest period recorded from teliospore formation to germination was 20 days, while 30 to 40 days was fairly common (Johnson, 1931).

In the spring, under favorable conditions, each cell of a viable teliospore can give rise to a typical promycelium, from which four *sporidia* are detached to be blown away by the wind. These sporidia cannot reinfect a cereal or grass host, and come to naught unless they fall on the surface of a barberry leaf. Here the germ tube enters the upper surface of the leaf, passing directly through the epidermis, and not through stomata. The resulting infections are of the type described under Symptoms, the pycnia appearing on the upper surface, while the æcia of the characteristic æcidium type follow upon the lower surface. It should be noted here that the mixing of pycniospores of opposite sex on the surface of pycnial spots is necessary for the formation of perfect æcia and æciospores. The separated æciospores are nearly globular, yellowish orange, with slightly roughened walls which appear almost smooth and vary in diameter from 14 to 26μ. These spores cannot reinfect the barberry, but their function is to carry the fungus back to its grass or cereal host. They can germinate at once, enter the stomata and establish a parasitic mycelium which will soon give rise to a crop of urediniospores.

It has been shown by Eriksson and Henning (1896) and Stakman and Levine (1919) that the urediniospores of the various biologic forms of the stem-rust pathogene differ considerably in size, and the latter have shown that the urediniospores become smaller when infection takes place under very unfavorable environmental conditions or when formed on a

fairly resistant host. Levine (1923) has recently determined the "limits of variation and the biometric constants for length and width of the æciospores, urediniospores and teliospores of a number of biologic forms," and concludes that "spore measurements can be employed as an additional aid in identifying the biologic forms of *P. graminis*, provided a sufficiently large number of spores are measured for both length and

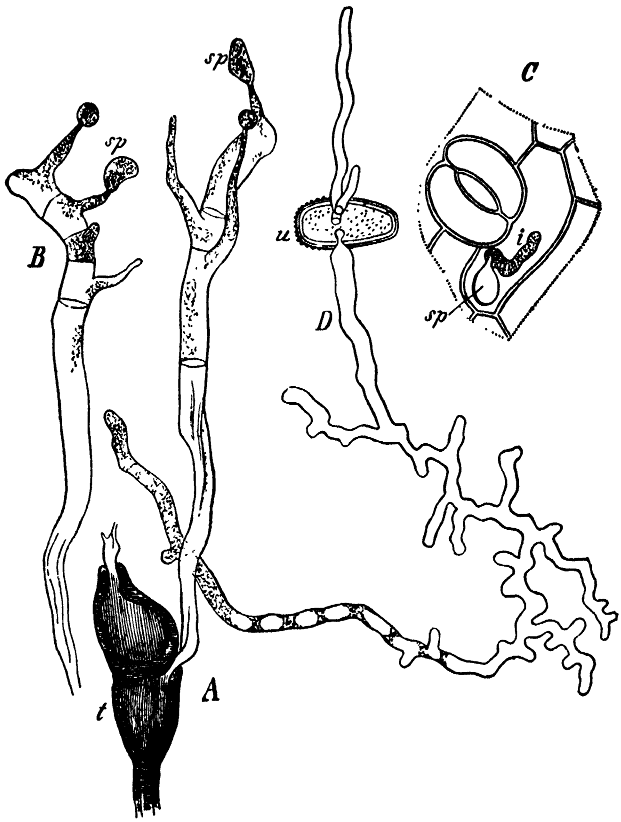

FIG. 221.—*Puccinia graminis.* *A*, germinating teliospore (*t*), with promycelium and sporidia (*sp*); *B*, a typical promycelium. (*After Tulasne*). *C*, a piece of epidermis from the under surface of a barberry leaf with germinating sporidium (*sp*) and infection thread (*i*); *D*, a germinating urediniospore. (*After De Bary.*)

width, and the spores studied are developed on congenial hosts under favorable conditions." He showed that

In general, the wheat rust (*P. graminis tritici*) has larger spores of each type than any other biologic form. The oat rust (*P. graminis avenæ*) occupies the second place; the rye rust (*P. graminis secalis*) third; and the timothy rust (*P. graminis phleipratensis*) the fourth. The redtop rust (*P. graminis agrostis*) has the smallest spores of all.

A later study of 8 physiological forms of *P. graminis tritici* showed them to be as distinct, morphologically, as *tritici* is from *avenæ* or *secalis*

(Levine, 1928). Waterhouse (1930) found significant morphological differences in a study on 13 physiological forms.

In some heterœcious rusts the alternate host is absolutely essential for the perpetuation of the pathogene, as is illustrated by the cedar rust of apples. In such a case the elimination of one of the hosts interrupts the life cycle and hence the fungus cannot develop. In stem rust, however, the pathogene is not so completely dependent on the presence of the alternate host, the barberry. The æcial stage on the barberry cannot be formed unless susceptible grasses or cereals are near at hand, but the pathogene can survive under certain conditions on the cereal host when barberries are absent or when the æcial stage is omitted. When barberries are present and the æcial stage appears on them, they are important agents in increasing the severity of stem rust, and are believed to be the most important cause of epiphytotics of stem rust. When the barberry stage is not formed there are several possible explanations for the presence of rust in an environment: (1) from urediniospores which have overwintered on volunteer cereals or wild grasses; (2) from wind transport of urediniospores from southern regions to more northern localities; and (3) by persistence on or in the seed.

It was at one time reported that urediniospores were able to live over the winter as far north as Madison, Wis., and St. Paul, Minn. Freeman and Johnson (1911) obtained as high as 40 per cent germination of overwintered urediniospores on Apr. 15. According to these workers, from Kansas and Ohio southward not only do a high per cent of the spores remain viable, but fresh spores can be found at any time during the winter. The opinion has been expressed that

> Where snow remains throughout the winter, preventing alternate freezing and thawing of material thus covered, the wintering of the urediniospore is, perhaps, facilitated. Indeed, it is very probable that the urediniospore survives the winter more easily in the North where snow is continuous during the winter than in localities where snow covers the ground only at intermittent periods (Freeman and Johnson, 1911).

According to Stakman, the importance of overwintering urediniospores as a source of the first spring infections has been overemphasized. He states that:

> In hundreds of experiments,[1] however, we have never been able to demonstrate that the urediniospores overwintered in the upper half of the Mississippi Valley. If they do, they do so so rarely as to be of no practical importance. The old results were undoubtedly correct as far as they went, but most of them did not go far enough. They found that the spores retained their viability until early spring, but that is just when they lose it. Spores frequently are viable until some time in March and even until early in April, but then they die quickly

[1] From letter from E. C. Stakman, June, 1925.

or germinate in the first rains of the spring when there is nothing for them to infect and are then, of course, harmless.

According to Lambert (1929), the overwintering of urediniospores is limited to regions where temperature and rainfall favor successive development, alternate freezing and thawing being a limiting factor. The wind dissemination up the Mississippi Valley the last 10 days of May and the first 10 days of June, when stem rust is at its height in Texas is believed to be capable of carrying rust to the spring-wheat area in 3 days' time. The movement of stem rust from southern to northern areas has been shown by other investigators to hold true for European countries.

It has recently been emphasized that stem rust lives over on volunteer wheat in Australia, a country in which the barberry stage is either unknown or rare (Waterhouse, 1920). More recently it has been shown that the power to infect the barberry has not been lost in Australia, as was formerly supposed (Waterhouse, 1929*b*).

As bearing on the possibilities of wind transport it may be noted that the urediniospores are resistant to desiccation and to exposure to light. Bolley obtained a germination of 8 to 15 per cent after their exposure on a watch glass for 21 days. The spores of some species have been shown to retain their viability after lying in herbarium specimens for nearly a year. The length of life of the urediniospores varies according to the temperatures and the relative humidity of the air. It has been shown (Peltier, 1922) that high temperatures lower the per cent of germination and shorten the viable period, while at low temperatures the opposite is the rule. At high relative humidities, depending on the temperature, the per cent of germination is low and the period of viability short. Medium humidity at a given temperature gives the longest viability. Form 21 at 49 per cent relative humidity at 5°C. gave 30 per cent germination at the end of a year (Peltier, 1925).

Their resistance shows that they could be carried long distances and still be capable of causing infections. Klebahn (1904), Freeman and Johnson (1911) and Stakman *et al.* (1923) have shown the prevalence of urediniospores in the air in rust regions by means of spore traps, and by means of spore traps on aeroplanes the last (1923) has shown the prevalence of the spores of rusts and other fungi in the upper air. There can be no doubt that the interchange of spores between localities is an important means of spreading the rust.

The telia of *P. graminis* have been found in the pericarp of wheat and oat kernels. This location of the rust pathogene was noted in this country by Pritchard (1911) and the possibility of such infected seed carrying the fungus to the new crop has been recently investigated by Hungerford (1920). From large numbers of samples collected from the rust epiphytotic of 1916, the largest per cent of infected seed was only about 1 per cent of the total. Under carefully controlled conditions no

rust appeared on plants grown from seed covered with viable urediniospores or bearing sori in the pericarp. At present the opinion prevails that stem rust is not transmitted from one wheat crop to the next by means of either contaminated or infected seed, bearing spores or dormant mycelium.

Brief reference may be made at this point to the *Mycoplasm Theory* of Eriksson (1897 and later). This theory recognized, "besides the well-known vegetative mycelium, another vegetative stage, when the fungus exists in the cells of the host plant as a formless plasma body, a sort of plasmodium, symbiotically fused with the protoplasm of the cells, and forming together with these a *mycoplasm*." This mycoplasm may be in the seed or other parts and under favorable conditions "the fungus forces its way out of the symbiotic complex, penetrates the walls of the cells and develops an intercellular mycelium." This theory has been entirely discredited by the work of Ward (1903) and others but stands as an illustration of how a preconceived notion and a misinterpretation of observations led to the "wearisome persistence in an inherently improbable hypothesis."

Predisposing Factors.—It is a matter of record that various portions of the world have been visited at various times with epiphytotics of stem rust of unusual severity; also that rust normally occurs in severe form every year in some sections, while in others it is normally present only in traces. These variations in severity must depend largely on climatic factors, with the barberry playing an important rôle in some regions. In order that an epiphytotic may occur, the following conditions must be fulfilled: (1) a supply of rust spores on the growing grain to give the disease a start; (2) temperature and moisture conditions favorable for germination and infection; and (3) a susceptible condition of the grain at the time spores are being generally disseminated. The first condition may be supplied by æciospores from barberries, wind-blown urediniospores or overwintering urediniospores, one or several of these sources generally existing. Infection is favored by moderately cool temperatures, abundance of dew, and humid, cloudy or misty days, while sudden showers followed by rapid clearing and evaporation of moisture are unfavorable. It has been claimed that the most susceptible period in the life of the wheat plant is from the time when the head emerges from the boot to the time it is in full bloom, thus giving a *critical period* of about 10 days for any one locality. According to Stakman, this is an apparent susceptibility only, the more numerous infections at this time being due rather to a greater abundance of inoculum.

In explaining the epiphytotic of 1904, it may be noted that the excess of precipitation over normal was greater in 1905 than in 1903 or 1904, so that, based on rainfall alone, 1905 should have had the most rust. In 1904, the temperatures were subnormal during the *critical period*, with a

general average of 2.67° below normal for Nebraska, Iowa, Minnesota, Wisconsin, South Dakota and North Dakota. These low temperatures, aside from favoring infection, retarded the growth of the crop, prolonged and delayed the period of susceptibility and gave time for the development of increased numbers of urediniospores. The importance of temperature and rainfall in producing an epiphytotic of stem rust has been emphasized more recently (Levine, 1928; Stakman and Lambert, 1928). An average temperature of 64°F. or higher with absence of drought conditions (at least 2 inches of rain) during the two summer months is favorable to the appearance of severe infections, while at temperatures below 60 to 61°F. or with drought conditions, little rust will appear.

Rust is increased in severity by heavy applications of nitrogenous fertilizers (Stakman and Aamodt, 1924; Gassner, and Hassebrauk, 1931), not by increasing susceptibility but by the increased density of stand and delayed maturity. The latter investigators have shown that potash fertilizers increase resistance but may result in reduced yields. Any factors which increase density of growth, such as heavy seeding or an undergrowth of weeds which hold moisture and exclude the sunlight, increase the severity of rust. Spring wheat generally suffers more from rust than winter wheat, and late-seeded spring wheat more than early-seeded. The slow-maturing cereal will be the most injured by rust, the rapid-growing the least, primarily because the rapid-growing variety reaches the critical period (or maturing) before rust has had time to reach its maximum of prevalence. It is stated that the epiphytotic of 1916 offered many illustrations of the value of early seeding or the importance of early-maturing varieties in escaping rust. For example, in Canada, Prelude wheat, ripening 10 to 15 days before Marquis, almost entirely escaped rust injury (Bracken, 1917). It is well known that 60-day oats with other quick-maturing varieties escape rust when other varieties with a longer period of growth suffer severely.

Varietal Resistance.—It has long been known that varieties of wheat show marked differences in their susceptibility to rust, and it has also been observed that *a variety apparently resistant in one locality may be severely rusted in another locality*, or that a variety may rust one season but not in another in the same locality. This behavior can now be explained by the existence of biological strains in one locality that are absent from another. For example, Rerraf, a variety very resistant in Australia, breaks down completely in North Dakota. The studies of Ward (1905) and others have led to the conclusion that resistance is not dependent upon anatomical peculiarities, but is physiological, that is, depends on physicochemical properties of the living substance, protoplasm. This is supported by the retardation of uredospore germination by filtrates of resistant varieties (Newton, *et al.*, 1929; Ezekiel, 1930). The idea is advanced that physiologic resistance is due to the liberation of phenolic compounds by the

host cells (Newton and Anderson, 1929). The basis for this theory is the reduction of infections of susceptible varieties by injections of phenolic compounds and the correlation between the natural phenolic content of a variety and its resistance. Acidity of the cell sap does not affect resistance (Hurd, 1924). In addition to fundamental protoplasmic resistance, wheat varieties may possess other means of defense, such as structural peculiarities which diminish the number of infections or restrict the growth of the mycelium after it has become established. Hursh (1924) and Stakman *et al.* (1925) point out that the relative amounts of chlorenchyma and sclerenchyma in the cortex affect rust development, since sclerenchyma constitutes a mechanical restriction on the growth of the mycelium. In addition to physiological and morphological resistance "functional" resistance has been offered as another type (Hart, 1929). This functional resistance is due to the daily rhythm of the stomata. On resistant hosts they open slowly and remain open only a short time, while in very susceptible varieties they open very soon after sunrise and remain open most of the day. This theory has not been substantiated by later studies (Peterson, 1931). Rust spores germinate on susceptible and resistant varieties in the same way and enter the stomata, but in resistant varieties the later development is modified according to the degree of resistance. In cases of high resistance or immunity there is a killing of the host cells adjacent to the substomatal vesicle, the fungus is unable to establish haustorial connections and soon perishes. In lesser degrees of resistance the killing of host cells may be delayed and a weak development of mycelium may give rise to small uredinia and undersized urediniospores (Stakman, 1914; Allen, 1923). The result is either sterile "flecks" or uredinia surrounded by hypersensitive areas of dead cells, although in three resistant varieties studied by Melchers and Parker (1922) "flecks are very rarely visible, and in no instance have even the most minute uredinia been observed."

In their study of 37 biological forms of *P. graminis* on wheat, Stakman and Levine (1922) recognized the following groups: (1) *immune*—no uredinia, flecks usually present, but sometimes not evident; (2) *very resistant*—uredinia minute and isolated, surrounded by distinct hypersensitive areas; (3) *moderately resistant*—small to medium-sized uredinia, with necrotic circle or halos, or sometimes in sightly chlorotic islands; (4) *moderately susceptible*—uredinia medium, coalescence frequent, no hypersensitive circles, but sometimes chlorotic areas; (5) *very susceptible*—uredinia large, numerous and confluent, entire absence of true hypersensitiveness, but with chlorosis under unfavorable conditions; (6) *heterogeneous*—uredinia very variable, all types and degrees on the same blade. Of the 12 differential hosts used, none were immune to the entire 37 biological forms, but Kanred was immune to 11. Mindum, Arnautka and Speltz Marz were very resistant or moderately resistant to a con-

siderable number of strains, while Vernal Emmer and Khapli Emmer were very resistant to the majority of the biological forms.

In testing about 130 varieties of winter and spring wheat, Melchers and Parker (1922) found that all varieties of winter wheat were susceptible except Kanred, P1066 and P1068. Black Persian was the only spring wheat of the *Triticum vulgare* group found to be resistant. Later studies, however, have shown Kota (Clark *et al.*, 1926) and Webster (Stakman *et al.*, 1925) to be very resistant, the latter resistant to more physiological forms than any other wheat. Kanred has the following additional desirable characters as tested in Kansas: (1) a yield of 3 to 5 bushels more than either Turkey or Kharkof; (2) it ripens earlier, escaping damage from drought and hot winds; (3) it seems to be more winter hardy than other varieties; (4) it is more resistant to smut than many other varieties. It is being rapidly established in many hard-winter-wheat areas and also has a potential value as a parent in breeding new varieties for rust resistance. Varieties of oats also show variable resistance to stem rust. Parker (1918) reports tests of more than 120 strains. "Unquestionable resistance to stem rust was present in two varieties, White Tartarian and Raukura Rustproof."

Prevention or Control.—In considering the prevention of stem rust it may be noted, first, that the disease is not seed-borne, hence seed disinfection is of no value. The type of the crop would make protection by spraying impractical, even if effective, but numerous tests made by Galloway and others have shown but little benefit from spraying. More recent trials with sulphur dusting (Bailey and Greaney, 1928; Lambert and Stakman, 1929) have given a fair control, but the adoption for farm practice is unlikely.

In areas in which rust injury may be expected, attention should be given to the selection of early-maturing varieties, seeding early rather than late, planting winter wheat wherever it is hardy, the avoidance of low, poorly drained sites or the excessive applications of nitrogenous fertilizers (see Predisposing Factors, p. 786).

Main reliance at the present is being placed on one or both of the two following measures: (1) the eradication of barberries, whether growing as hedges or ornamentals or run wild; (2) the selection and breeding of resistant or immune varieties. The value of barberry eradication in the prevention of epiphytotics of rust is well illustrated by the experience in Denmark and in England (Stakman, 1923). England has eradicated the barberries without the aid of law, and black rust is almost a thing of the past, but in adjacent Wales, where barberries are still abundant, rust losses are serious each year. In Denmark, when barberries were still numerous, there were severe outbreaks of rust in 1893, 1895, 1896, 1897, 1900 and 1901, but in 1904 an effective barberry law was put into operation, with the result that no general outbreaks of stem rust have since

occurred. In regions in which the cluster-cup stage of rust develops on the barberry the elimination of this alternate host is the first step in stem-rust control. The barberry should not, however, be branded as a general menace, since in many environments it plays no part in the life of the rust pathogene (see Geographic Distribution, p. 776). This absence of the barberry stage is probably due to the inability of the teliospores to live through the hot and sometimes also dry summers, since they have been shown to lose their vitality if kept for several months at 35°C. The barberry eradication campaign is complicated by the fact that this shrub has run wild in many sections of the eradication area, and by its ability to sprout up again from the roots when cut off or dug out. Chemical treatments (Thompson, 1923; Thompson and Robbins, 1926) are being recommended: (1) 10 to 20 pounds of common salt piled over the crown of a bush; or (2) 2 gallons of sodium arsenite solution (1 gallon of concentrated sodium arsenite to 40 to 50 gallons of water) poured on the crown, the salt treatment giving the best results. In 1923 over 5,000,000 bushes had been removed in the eradication area from Ohio to Montana since the beginning of the campaign.

The Japanese or Dwarf barberry (*Berberis thunbergii*) does not rust, and is safe for ornamental planting. It can be distinguished from the dangerous common barberry (*B. vulgaris*) by the following comparison:

B. vulgaris	*B. thunbergii*
Berries: In bunches like currants.	Single or in twos like gooseberries.
Leaves: Margin spiny toothed.	Margin smooth.
Spines: Usually in groups of three.	Usually single.

B. canadensis and *B. fendleri* are two native varieties of restricted range that carry stem rust, the former mainly in the mountains of West Virginia, Virginia, North Carolina, the latter in southern Colorado. Only the former is of importance.

Mahonia or Oregon grape, closely related to the barberry, is represented by two species. The tall Mahonia (*M. aquifolium*), wild in the Pacific Northwest and cultivated elsewhere, may rust slightly, and hence is not recommended for planting in rust zones. The æcial stage of stem rust has never been found on this species in the Pacific Northwest but it is sometimes attacked by another species, *Puccinia fendleri*. The low training form (*M. repens*), which occurs in the western portion of the eradication area and further west, never develops the æcia of stem rust, but it is commonly affected by *P. mirabilissima*, a species of no economic importance.

Some progress has been made in the selection and breeding of resistant or immune strains, but this work is complicated by the large number of biological forms of stem rust and their variable occurrence in different environments. Crosses have been obtained by Hayes *et al.* (1920) of

common, durum and emmer types more resistant than the resistant durum parents and later work by Aamodt and others (1923) has shown "that varieties of common wheat may be produced synthetically which will be resistant to a large number of the biologic forms of stem rust." The breeding of rust-resistant varieties has opened an almost endless field, and numerous plant breeders have made important contributions to this phase of the problem.

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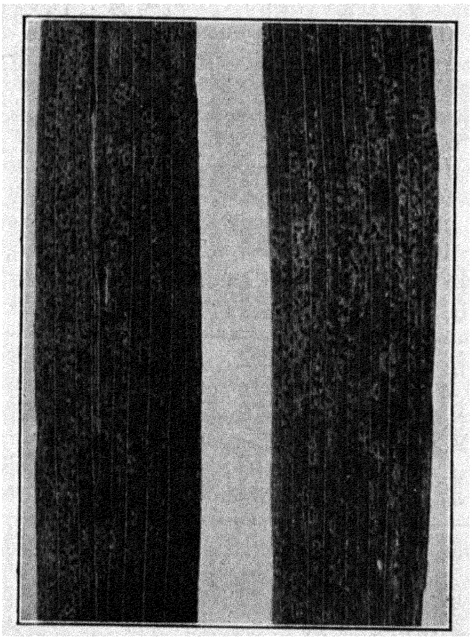

FIG. 222.—Leaves of wheat showing uredinia of *Puccinia triticina* and the hypersensitive areas caused by this rust.

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Tabular Comparison of Cereal Rusts

	Stem rust (*Puccinia graminis* Pers.; *Dicæoma poculiforme* (Jacq.) Kuntze)	Yellow stripe rust (*Puccinia glumarum* Erik.; *Dicæoma glumarum* (Sch.) A. & F.)	Orange leaf rust (*Puccinia triticina* Erik.; *Dicæoma clematidis* (DC.) Arth.)	Crown rust of oats (*Puccinia coronata* Corda, *Dicæoma rhamni* (Pers.) Kuntze)	Brown leaf rust of rye (*Puccinia dispersa* Erik.; *Dicæoma asperifolii* (Pers.) Kuntze)	Dwarf leaf rust of barley (*Puccinia simplex* (Koern.) E. & H.; *Dicæoma anomalum* (Rostr.) A. & F.)
Cereal hosts II, III	Wheat, spelt, emmer, einkorn, oats, barley, rye and grasses	Wheat, spelt, emmer, barley, rye and 33 grasses	Wheat, spelt and grasses	Oats and various grasses	Rye	Barley
Alternate host 0, I	Barberry (*Berberis spp.*, *Mahonia aquifolium*)	None known	Meadow rue (*Thalictrum spp.*) and other Ranunculaceæ	Buckthorn (*Rhamnus spp.*)	Borage (*Anchusa arvensis* and *A. officinalis*) in Europe	*Ornithogallum tenuifolium* and *O. umbellatum* in Europe only
Location of sori	Stems and leaf sheaths; on glumes and leaves when severe	Most numerous on leaf blades; also on sheaths, stems and glumes	Leaves, rarely on other parts	Leaves, sori amphigenous	Leaves, uredinia epiphyllous, telia hypophyllous	Leaves, sori amphigenous
II Sori: Form	Linear, large; scattered or coalescing in long lines	Oval, small, arranged in evident parallel lines or streaks	Oval or round, larger than in stripe rust; scattered	Scattered, elliptic, 0.5 to 1 millimeter	Oblong, scattered, 0.5 to 1 millimeter, early naked	Scattered, oblong or linear, 0.5 to 1 millimeter, early naked
Color	Yellowish brown, becoming darker	Lemon yellow	Bright orange when fresh, darker with age	Brownish yellow	Reddish to cinnamon brown	Cinnamon brown
III Sori: Form	Like II, open early	Like II, but flat and covered; mostly on under surface	Like II, but flat and covered; mostly on under surface, sometimes absent	Scattered, oblong, long covered by epidermis	Broadly oblong, 0.5 to 1 millimeter, long covered by epidermis	Scattered elliptic or oblong 0.2 to 1 millimeter, long covered by epidermis
Color	Black	Dull black	Dull black	Blackish brown	Blackish brown	Dark blackish gray
Urediniospores	Ovate oblong to ellipsoid, 17 to 45 by 14 to 22μ; four germ pores, equatorial; wall brownish	Nearly globular, 23 to 35 by 20 to 35μ; six to 10 germ pores, scattered; wall colorless	Subglobose, 20 to 28 by 18 to 21μ; four to six scattered germ pores; wall brownish	Globoid or broadly ellipsoid, 16 to 20 by 18 to 24μ; wall pale yellow, six to eight scattered pores	Globoid or broadly ellipsoid 18 to 24 by 21 to 29μ; wall golden yellow or pale cinnamon brown, six scattered pores	Broadly ellipsoid, 16 to 24 by 22 to 29μ; wall yellowish, eight to ten scattered pores
Teliospores	Rounded or pointed above, rarely truncate, 35 to 65 by 11 to 22μ; pedicel elongated, tinted	Rounded or flattened above, generally truncate, 35 to 63 by 12 to 20μ, pedicel short, concolorous with wall	Rounded or flattened above, generally truncate, 39 to 57 by 15 to 18μ; pedicel short, colored	Clavate oblong or cylindric, 13 to 19 by 30 to 67μ, with crown of three to ten digitate projections	Oblong clavate or cylindric, 15 to 21 by 37 to 61μ, truncate or rounded above, narrowed below	Angularly oblong or clavate, 18 to 25 by 39 to 58μ, truncate, rounded or obtuse above, narrowed below; mesospores abundant

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APPLE RUST

Gymnosporangium juniperi-virginianæ Schw.

The common rust of apples east of the Rocky Mountains, which is frequently spoken of as cedar rust or the cedar-rust disease of apples, has become so widespread in that section as to give nearly as much concern as apple scab or fire blight. In the most favorable localities it has developed to such an extent as seriously to threaten the productiveness of apple orchards. The fungus which causes the disease passes a part of its life cycle upon the leaves, fruit and twigs of the apple, producing the characteristic *rust spots*, while upon the common cedar (*Juniperus virginiana*) it causes the formation of the well-known "cedar apples" or cedar galls, thought by some people to develop the "cedar flowers."

History.—Although the organism responsible for this disease was first described by Schweinitz in 1822 and was studied in subsequent years by mycologists, it was not until many years later that the trouble became sufficiently serious to call for protective measures. Control measures were considered by pathologists in a number of the eastern states about the same time. In 1889 Galloway of the U. S. Department of Agriculture reported on spraying experiments conducted at Vineland, N. J., in 1888, while Halsted in the same year considered the possibility of some varieties of cultivated apples being more susceptible than others. Jones, working in Vermont, in 1889 sprayed apple trees, for cedar rust, with ammoniacal copper carbonate and reported the results 2 years later. These first experiments showed little benefit from spraying, but work which he did a year later gave fairly good results. He concluded, however, that spraying was not a very practical method of control. In 1892 Pammel reported spraying the wild crab for cedar rust, but the disease was not prevalent on apples in Iowa at that time. The first recommendation for the control of the apple rust by destroying the cedar trees was made by Jones in 1893, who reported magical results from the creation of a cedar-free zone for a radius of 1 mile around

an orchard that had suffered severely. By 1901 the disease must have been fairly prevalent in the South, as Austin reported on spraying experiments in Alabama. The disease was first reported to affect apples in Wisconsin, Iowa, Minnesota and Nebraska in 1903, although the fungus had long been known on its other hosts in that territory. According to Pammel, there was a severe outbreak of the apple rust in Iowa in the summer of 1904 and adjacent regions suffered to much the same extent. In 1905 Emerson reported on successful spraying experiments carried out at the Nebraska Experiment Station. The following year the author began a special study of the rust problem in the same state, giving particular attention to the life history of the causal fungus, and to the possibility of controlling the disease on the alternate host, the common cedar, the results appearing in 1908. Between 1910 and 1915 apple rust was the subject of special study in New York by Stewart (1910), in Wisconsin by Jones and Bartholomew (1911–1915), in Alabama by Lloyd (1911), in Nebraska by Coons (1912), in West Virginia by Giddings and assistants (1911, 1915, 1918), in Virginia by Reed and co-workers (1912, 1914, 1915), in North Carolina by Fulton (1913), and in Virginia by Fromme and others (1918–1920). Extensive technical studies have been published by Reed and Crabill in Virginia (1915) and Giddings and Berg in West Virginia (1915 and 1918). The volume of work reported by Experiment Station workers in recent years may be taken as some indication of the severity and importance of apple rust as an orchard disease.

In Virginia and West Virginia, the severe epidemics of 1910 and 1912 served as a special stimulus to the study of the cedar-rust problem in both field and laboratory. During 1913–1914 the West Virginia State Crop Pest Commission began active work on the destruction of cedars as a means of controlling rust, while a special Cedar Rust Law was enacted in Virginia and became effective in 1914. In those counties in which the law has been put into operation most excellent results have been obtained by the destruction of cedar trees, although many protests have been made by owners of cedars (Brooke, 1930).

Geographic Distribution.—According to Giddings, the common rust of apples is widely distributed throughout the eastern and central portions of the United States from Maine to Florida and westward to South Dakota, Nebraska, Kansas and Oklahoma. It is recorded also from Ontario, Canada and from Alaska, but seems to be absent from the mountain states and from the Pacific Coast section. The disease seems to have attained its greatest severity in the prairie states of the central Mississippi Valley and in the commercial apple districts of Virginia and West Virginia. The unusual development of the disease in such states as Iowa and Nebraska seems to have been due to two factors: first, the fairly common practice of planting the cedar tree as a windbreak around orchards and as ornamental trees for the home grounds; second, to the frequent use of a very susceptible variety, the Wealthy. In Virginia and West Virginia, the common cedar is very much at home, as its name indicates (*Juniperus virginiana*), and is frequent along the roadsides even in the commercial apple districts. This natural distribution of the cedar and its ready reproduction, coupled with the extensive plantings of another very susceptible variety of apple, the York Imperial, gave ideal conditions and "cedar rust thrived and waxed fat and multiplied" (Fromme, 1918).

Symptoms and Effects on Apple Trees.—The rust affects leaves, fruits and more rarely the young, tender twigs. Leaf attacks are common throughout the range of the disease, and in some localities constitute the main phase of the trouble. The rust first shows on the upper surface of the leaves as pale-yellow spots about the size of a pinhead, being first in evidence about 10 days after the "cedar apples" on adjacent cedar

trees have been in the gelatinous condition. The spots increase in size and assume a deeper color, finally becoming orange-colored, frequently with reddish borders. Minute pustules appear in the center of the spots and these show later as small black specks. The leaf tissue beneath the spots soon begins to swell up in case of well-separated spots and produces a cushion or blister ¼ to ½ inch in diameter. Minute tubular projections, the cluster cups, appear on these cushions, and when mature (late June and July) split open and recurve their walls, so that each has a stellate appearance. In the bottom of each open cup a mass of brown, powdery spores is produced. While the above may be considered typical for leaf infections, the attendant symptoms may vary. The two principal deviations are due: (1) to numerous infections in susceptible varieties; (2) to aborted infections on resistant varieties. The size of the spots will vary with the number of infections per leaf, which may be as high as 200 to 300 in the case of susceptible varieties standing close to cedar trees. With a large number of infections the spots remain small and coalesce, and in some cases the leaves may turn yellow and fall before the cluster cups are formed. Heavily infected leaves may also be more or less curled or rolled. In certain varieties infections may result in the formation of circular, brown spots of dead tissue ⅛ inch or slightly more in diameter, which show minute black pustules in the center. These infections suggest leaf spots due to imperfect fungi, the central pycnia resembling pycnidia. These aborted infections never make any further development. Sometimes the rust spots remain minute and undeveloped for the entire season, at times not even being able to form the pycnia. The aborted infections are found either on resistant varieties or on susceptible varieties which have been infected after the leaves are nearly mature.

Rust lesions may also appear on the young twigs of very susceptible varieties, but they are rare in comparison with leaf and fruit lesions. Affected twigs will show slightly enlarged cushions covered with the characteristic cluster cups. They may appear either at the nodes and surround and involve a bud, or they may be located in internodal areas; Smiths' Cider is reported (Reed and Crabill, 1915) as showing seriously disease twigs, Hopkins (1922) has recorded a case of heavy twig infection on the Yellow Bellflower, and more recently (Young, 1927) has reported severe injury to 1-year-old Ada Red trees in Arkansas, with the production in many cases of deep-seated, girdling cankers that caused a killing of the distal portions. Twig infections were also fairly common on mature trees of the same variety.

In susceptible varieties of apples the young fruits may be very generally infected. The lesions may be localized at the calyx end of the fruit and involve the calyx lobes, but they may also be on the side or at the stem end or general over the entire surface. Young infections will show

characters similar to the upper surface of leaf lesions, but later cluster cups will break through the same lesions, generally appearing in a ring around the central pycnial pustules. There may also be a greenish or yellowish discoloration of the flesh extending to the core and quite often pycniospores may be found in the core. Jonathan, York and Ben Davis are particularly susceptible to fruit infection. Slight infections

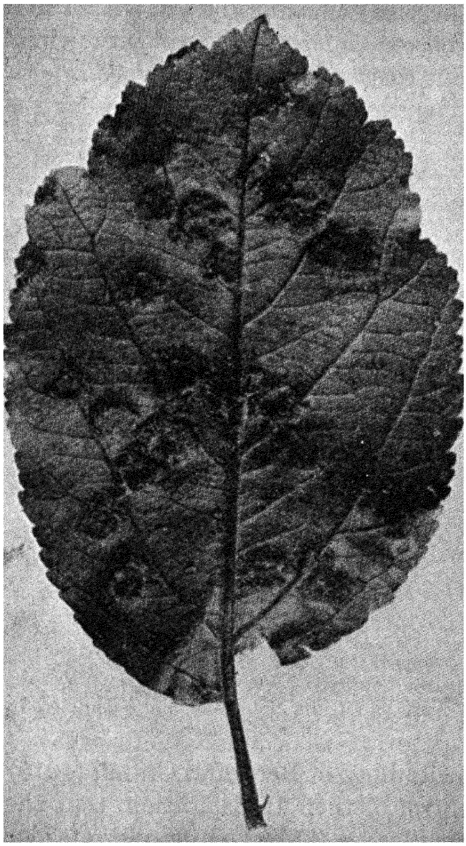

FIG. 223.—Mature æcia on the under surface of an apple leaf.

of the fruit have only a disfiguring effect, while in more severe development, deforming and atrophy may result.

The injury to the apple from rust is due to: (1) leaf infections and the resultant defoliation; (2) dwarfing and reduction of quality of the fruit. Foliage of susceptible trees standing adjacent to cedars may bear so many lesions as to appear a pronounced yellow even from a distance, and in such cases there will be an early defoliation. It has been shown that the leaf fall is proportional to the number of infections per leaf. Giddings and Berg report that in 1914 York Imperial leaves with 10 or

more rust lesions showed a dropping of 55 per cent before Sept. 1. The spotting and the dropping of the leaves constitute a severe drain on the vitality of the trees, and therefore they go into their winter rest in a weakened condition. The effect on the fruit may be direct from localized infections, but "it is believed that leaf infection is a far more important factor than fruit infection in determining fruit size" (Giddings, 1918). The effect of severe leaf infections on fruit setting and size may even be carried over into the year following the attack. The fruits showing rust lesions frequently become infected with rot-producing fungi which cause still further loss, but rust spots do not increase in either size or number during storage. The physiological effect of cedar rust on the apple is

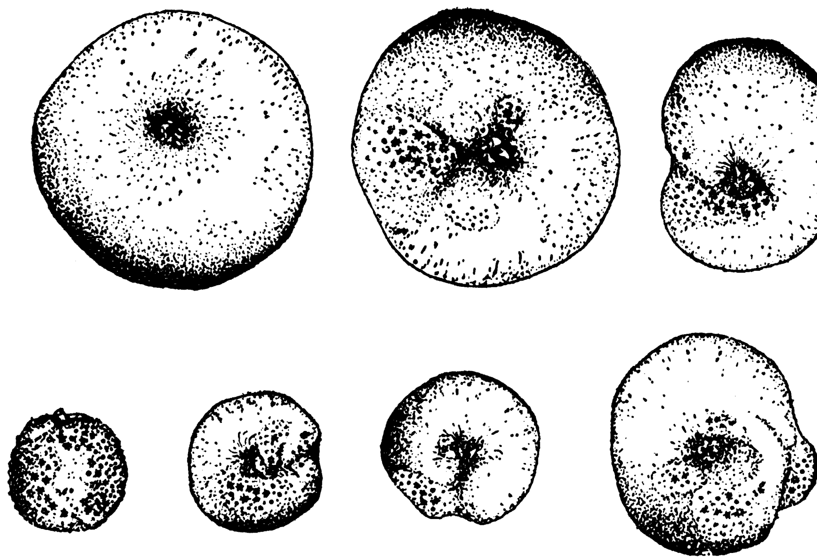

FIG. 224.—A normal fruit and fruits deformed and atrophied by apple rust. (*Drawn from a photograph.*)

emphasized by the findings of Reed and Crabill (1915) that rusted leaves appear to lose control of their transpiration, that the process of photosynthesis in diseased leaves is very materially retarded, and that respiration is more rapid in the diseased than in the healthy foliage.

Losses from cedar rust have been especially heavy in Virginia, West Virginia, Iowa and Nebraska, but evident, though less serious, in other states. Fromme (1918), writing of conditions in Virginia, says:

The effect of cedar rust on the York is quite different from that on Ben Davis. The attack is chiefly on the foliage, which falls early, leaving half-nourished, undersized apples. When the infection is heavy the loss is almost complete. Promising number ones are reduced to culls. The tree itself suffers severely. Growth is arrested, few fruit buds are formed and the next year's crop is sure to be a light one. A heavy cedar-rust infection thus means a loss of a large part of 2 years' crops.

The loss for Shenandoah County, Virginia, was estimated at $100,000 for 1917. Giddings and Neal (1912) reported that "the rust of the apple has been one of the most serious diseases found in many of the orchards of West Virginia during the past few years. The damage this year in one county is placed at $75,000." These illustrations will suffice to emphasize the importance of cedar rust as a factor in commercial orchard districts.

Symptoms and Effects on Cedar Trees.—The disease appears on the red cedar in the form of chocolate-brown, globular, subglobular or reniform, corky galls of varying sizes, from those scarcely 1/16 inch in diameter to others 2 inches across, scattered over the tree. The young galls are

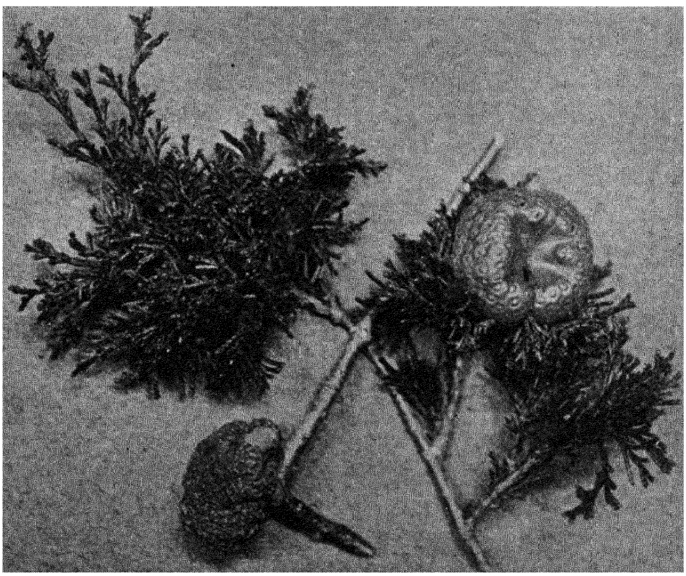

FIG. 225.—"Cedar apples" or galls (*Gymnosporangium juniperi-virginianæ*) on the common cedar. One at the right alive, the other an old gall of the previous season.

first evident in June as minute globular structures in the axils of leaves or slightly removed from this position on the leaves. They grow rapidly during the summer, and by fall have reached mature form and size. At this time they show circular depressions scattered over their surface. In April or May of the following season or during the first warm spring rains an elongated, gelatinous, orange-colored horn or projection is pushed out from each depression, and in this condition the larger galls have quite a flower-like appearance, which has suggested the popular name of "cedar flowers." The smallest galls may produce only one or two gelatinous horns, while large ones may form 300 or more. These gelatinous horns dry up and the old galls are left, finally as dark-brown or almost black, hard structures which may persist on the tree for some time. In midsummer a cedar tree which had been affected for a sufficient length of

time would show young galls just beginning to form, galls which had "bloomed" in the spring and some old galls of former seasons.

Cedar trees may show only a few galls, or they may be so heavily infected that their branches are bent with the weight of the "cedar apples." The injury to the cedar is of little concern in those localities in

FIG. 226.—A single large gall with completely expanded, gelatinous telia.

which this tree is common in the pastures, waste places and roadsides (Virginia and West Virginia), but in the plains country in which cedars have been used for windbreaks or for ornamental plantings they are frequently prized as highly as the apple trees. The amount of injury to cedar trees varies with the severity of the infections and the age of the trees. Observations in Nebraska (Heald, 1909) showed that trees from

20 to 30 years old were the most susceptible to infection and suffered the most from the presence of the parasite. In many places severely infected cedars were found to be dying and there was little doubt that the cedar rust was responsible for their destruction.

Etiology.—Cedar rust is due to one of the true rust fungi, *Gymnosporangium juniperi-virginianæ* Schw., a heterœcious pathogene, which passes a part of its complex life cycle on the apple and a part on the common red cedar (*Juniperus virginiana*), the proximity of the two hosts being essential to the perpetuation of the disease. In the earlier literature the pathogene appeared under the name of *G. macropus* Lk. When the stages on the two hosts were first observed and studied they were thought to be independent fungi, and the æcial stage on its pomaceous

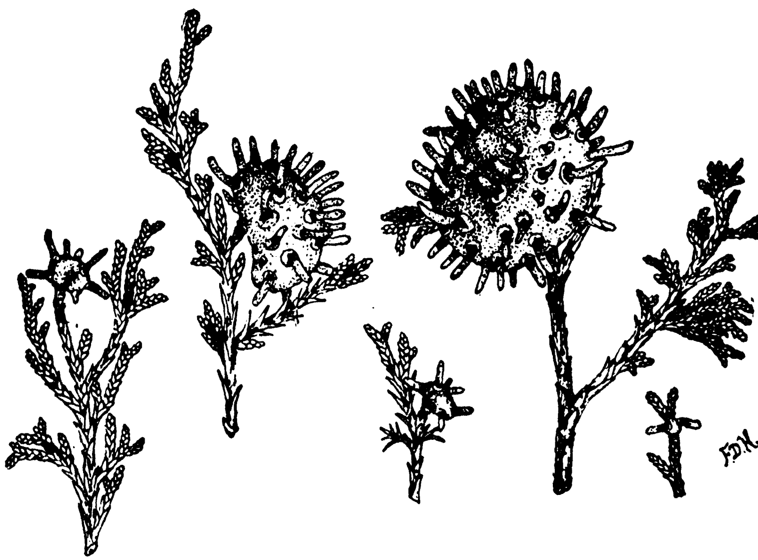

FIG. 227.—"Cedar apples" of various sizes just beginning to show telia. (*Natural size. Drawn from a photograph.*)

hosts was described as *Rœstelia pyrata* (Schw.) Thax. The first genetic connection between Gymnosporangium species and Rœstelia or æcidium forms was established by Oersted in 1865, and since that time all Gymnosporangia have been shown to have a Rœstelia stage.

Another species, *G. globosum* Farl., also parasitizing the red cedar, is the cause of apple and pear rust in the eastern United States, while the western rust (*G. blasdaleanum*) of the Pacific Coast alternates between the incense cedar (*Libocedrus decurrens* Tor.) and cultivated apples, pears, quinces and wild Pomeæ.

The following is a summary of the life cycle of the cedar-rust pathogene: (1) spore horns, or telial sori are produced from galls in the spring; (2) the teliospores embedded in these germinate during warm spring rains

and produce promycelia with secondary spores or sporidia; (3) as the humidity decreases a little (a drop of 10 per cent or more) the sporidia are forcibly abjointed and are blown away by the wind; (4) the sporidia falling on young leaves or fruits of the apple germinate and start infections; (5) after a period of incubation pycnia appear on the upper side of the leaf lesions, to be followed later by the æcia on the lower surface; (6) the æcia produce the æciospores, which fall out of the cups and are carried to the cedar, where they germinate and start infections; (7) the host responds by the formation of the characteristic galls which reach maturity in the late fall and are ready to form telial horns in the spring.

Compact masses of fungous tissue develop beneath the cortex of the gall depressions and early in March, April or May, depending on the latitude, the pressure of expanding "buffer" cells (Dodge, 1918; Stevens, 1930) causes the rupture of the cortex, and the telial column or sorus begins to emerge. The tentacle consists of the elongated gelatinous stalks of the two-celled teliospores, which are pushed out to the surface by the elongation of these stalks. The telia are golden yellow, cylindrical, acuminate and vary from less than ½ inch to more than 1 inch in length. The teliospores are oval to acuminate, frequently constricted at the cross-septum and 15 to 20 by 46 to 60μ. With the absorption of water from the spring rains the sori swell to enormous size and become more gelatinous and the teliospores germinate in situ. This may be repeated during periods of rainy weather, up until about June 1, the sori drying in the interim, so that there may be one to as many as six or even more periods of telial germination before the supply is exhausted, the number of periods depending upon the weather conditions. Each cell of a teliospore can grow out into a hypha-like structure, the *promycelium*, which under typical conditions becomes four-septate, leaving a sterile stalk cell and four fertile or basidial cells, each of which produces a secondary spore or *sporidium*, borne on a short lateral projection (sterigma). A teliospore thus produces eight sporidia. It has been estimated that a single gall 1¾ inches in diameter may produce the enormous total of 7,440,000,000 sporidia, which will emphasize the fact that a single cedar tree may be a menace to an entire orchard (Lloyd and Ridgway, 1911). Coons (1912) has shown that, as the telia dry following a rain period, the sporidia are forcibly abjointed from their sterigmata and are thus set free from the telia, after which they are readily carried away by the wind. The number which fall on apple foliage will depend upon the distance of this host from the cedar trees. Under favorable conditions the sporidia germinate by the formation of a germ tube which very soon penetrates the epidermis of the upper leaf surface and an infection results.[1] The period between

[1] In an article which the writer had no chance to edit this statement was changed to read, " . . . produce infections by the entrance of infection threads through stomata into the tissues of the host" (Heald, 1909).

sporidial dissemination and actual infection is very short, being reported as short as 6 hours. This behavior has a very important bearing on control, making it especially difficult to prevent infections by spraying.

After a period of incubation of 7 to 16 days, the rust becomes visible on the upper surface as pale-yellow spots about the size of a pinhead. The spots enlarge and assume a darker shade of yellow and in about 2 weeks little raised specks appear in the center, the openings of the flask-shaped pycnia embedded within the leaf tissue. These pycnia soon exude a thick orange-colored fluid, which contains the pycniospores.

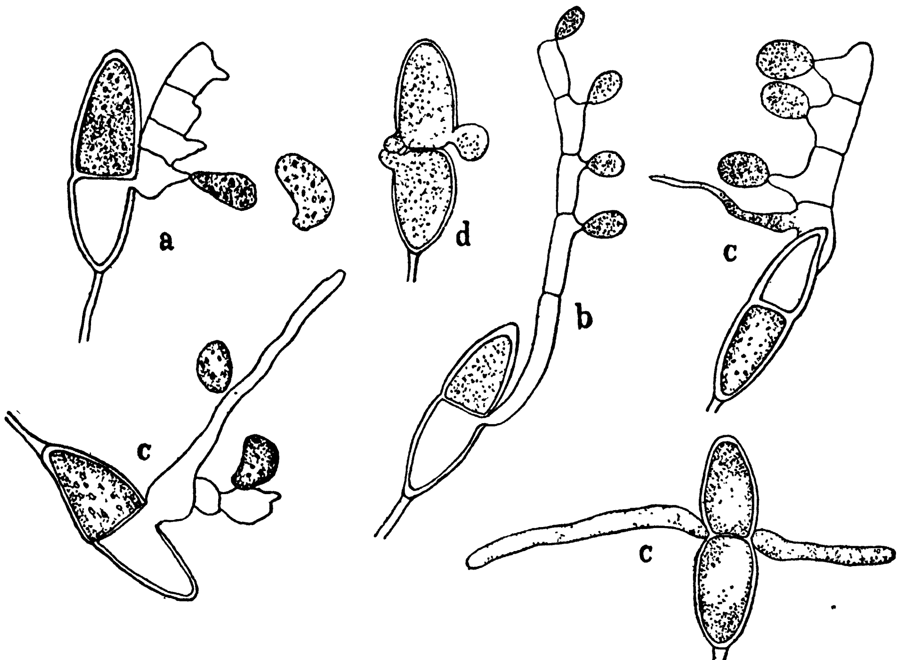

FIG. 228.—Germination of teliospores of *Gymnosporangium juniperi-virginianæ*. *a*, typical germination, compact form; *b*, typical germination, elongated type; *c*, promycelia showing deviation from the typical; *d*, teliospore showing direct production of sporidia.

With the cessation of pycniospore formation the pycnia appear as small black specks. The pycniospores probably play the same part as in other heterœcious rusts.

The mycelium of the rust fungus is intercellular but sends haustoria into the cells of its host. Its presence stimulates the invaded tissue and an excessive enlargement and multiplication of the spongy parenchyma cells result in the formation of the characteristic cushion or swelling that develops on the under side of a lesion. These swellings are at first unbroken, but soon (late June or July) the æcia which have been forming within break through the under epidermis, and protrude at first as brown papillæ. The outer coating or *peridium* then ruptures, gradually splitting

to the base as it elongates and during dry weather the segments or rays become recurved giving the open æcium a stellate appearance.

The peridium is composed of a single layer of cells which forms a covering for the æcidiospore chains within. The peridial cells are virtually modified æciospores and are produced by a differentiation of the apical æcidiospores at the top of the æcidium and of whole chains of æcidiospores on the sides of the æcidium (Reed and Crabill, 1915).

The æciospores are borne in chains within the peridium and are gradually set free as they mature. During the wet weather they are prevented from falling out by the closure of the peridial strands or segments, but during dry weather they are easily dislodged. The mature æciospores are

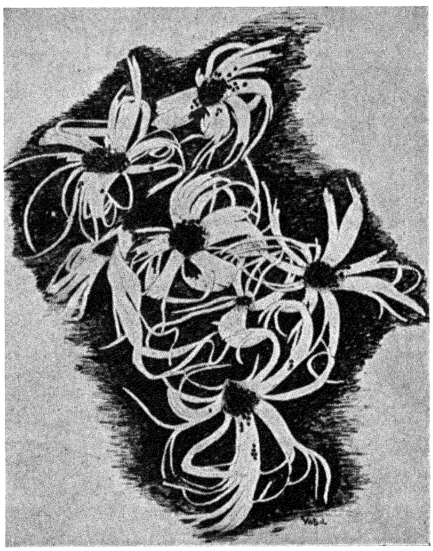

FIG. 229.—A group of æcia much enlarged. (*Photo of drawing by Mrs. Venus Pool McKay, Neb. Exp. Sta. Ann. Rept.* **22**, 1909.)

almost spherical, dark brown, with minutely pitted walls, somewhat darker than the granular cell contents. The younger and deeper lying æciospores are more or less polyhedral, due to pressure of the adjacent chains. The æciospores are apparently dependent upon the wind for their dissemination. If they reach susceptible portions of the cedar tree and other conditions are favorable, they may germinate and produce infections; otherwise they will perish. They germinate very poorly during the summer, but some are viable even as late as October, hence it is not known just when the actual infection of the cedar occurs. Two possibilities have been suggested: (1) that infections occur soon after dissemination of the spores; and (2) that the spores remain dormant until spring and germinate about the time growth starts in the cedar. Whichever happens, it is certain that the young galls do not make their

appearance until the June following the æciospore dissemination, which takes place in July, August and September.

The galls were at first supposed to originate as outgrowths from young stems, and this idea was supported by a number of investigations. Stewart (1915) from a histological study concluded that they originate as modified axillary buds, but Weimer (1917) has presented evidence that they originate as modified leaves. The most common point of origin is close to the base of the leaf on the upper side, hence superficial examination suggests axillary origin. The development of the gall does not start until the cedar starts growth in the spring. Growth then continues quite rapidly, and by late fall the galls have reached full size. Internal organization of the telia proceeds slowly and the surface depressions become more pronounced. The mature gall is a mingled mass of parenchyma and vascular tissue of the host and an intercellular mycelium of the pathogene surrounded on the outside by layers of brown cork cells. In early spring the overwintered galls are ready to produce the gelatinous sori with their crops of teliospores, and the life cycle will be repeated.

It was formerly supposed that the "cedar apples" matured in the fall originated from infections brought about by æciospores of the preceding spring, but the writer first showed (Heald, 1909) that the young galls appear before the cluster cups of that season have begun to shed spores, hence they must originate from æciospores of the preceding season. Even if the æciospores should lie dormant in the leaf axils during the winter period, it still means that the pathogene requires approximately 23 months from the time of æciospore dissemination to complete its life cycle.

The spraying of cedars Sept. 2 and later, which gave almost no reduction in the number of galls over unsprayed trees, and the marked reduction in infection secured from sprayings made in July and August, support the idea of infection following shortly after æciospore dissemination rather than being delayed until the following spring (Heald, 1909).

Conditions Which Influence Infection of Apples.—In order that rust may develop in an apple orchard there must be infected cedars not far distant, and then the amount of rust which appears will depend on: (1) the location of the trees with relation to the cedars; (2) the weather conditions which prevail at critical periods; and (3) the development of the apple foliage at the time of dissemination of the sporidia. According to Thaxter (1891), infection may take place when the nearest cedars are 8 miles distant, but it has been shown that a cedar-free zone of 1 mile around an orchard is usually sufficient to give commercial protection. Windbreaks close to orchards are favorably located because of proximity of the two hosts and because prevailing winds would carry the sporidia into the orchard. The elevation of cedar trees with reference to adjacent

orchards or the presence of intercepting barriers between cedars and apples will modify the severity of infections. Giddings and Berg (1915) state that:

> A rain lasting at least 2 or 3 hours is usually required to bring about spore production and discharge on cedar apples; the rust spores are usually discharged for only a few hours after a rain or between showers, when the humidity of the air drops somewhat; the cedar apples are particularly active in discharging spores after a prolonged dry spell, but they appear to be temporarily exhausted after two or three closely successive periods of discharge; and ordinarily a wind velocity of at least 3 or 4 miles per hour is necessary for a sufficient distribution of spores to cause any general infection.

The amount of rust will depend on the age of leaves at the time of sporidia dispersal, since only young leaves can be infected. Under some conditions sporidia may be disseminated before the leaves have developed sufficiently to become infected, while at other periods of spore dispersal some of the foliage may be too old to take the disease. The coincidence of a period of heavy sporidial dispersal with a period when many of the apple leaves are young will give maximum infection. Since sporidia live for only a few days, those disseminated previous to the appearance of the leaves will come to naught. "Penetration of the germ tubes takes place through the dorsal epidermis of the leaf, and it therefore seems probable that the increasing thickness of cell walls and cuticle is the factor which determines the period of possible infection" (Reed and Crabill, 1915).

Host Relations.—It seems probable that the cedar rust first occurred on the wild crab (*Pyrus coronaria*) in the southern and eastern United States and at first was confined to this host and the cedar. Its spread to apple orchards parallels the development of commercial orchards, although its appearance in Iowa orchards had not been noted previous to 1904, yet it had been observed on the local wild crab (*P. iowensis*). Early attempts to inoculate cultivated apples failed, and the final establishment of the disease in apple orchards is believed to be due to the extensive planting of very susceptible varieties, the Wealthy in the plains country of Iowa and Nebraska and the York Imperial in Virginia and West Virginia. The Rœstelia stages on pears, quinces, serviceberries and hawthorns belong to other species of Gymnosporangium.

Observations have been made in the various states on the comparative susceptibility of different varieties of apples. Giddings and Berg (1915) recognize the following groups: (1) susceptible; (2) moderately susceptible; (3) resistant; and (4) immune. Some varieties are listed differently in different states—for example, the Ben Davis, susceptible in Virginia and Iowa; moderately susceptible in West Virginia; and resistant in Nebraska, Massachusetts, Rhode Island and Delaware. Evidently resistance may be only apparent, since some varieties may escape

infection due to time of expansion of the foliage or its rapidity of development. There seems to be a general agreement that such valuable varieties as the Wealthy, York Imperial and Jonathan are very susceptible. Varieties like Baldwin, Stayman, Winesap, Arkansas Black and Yellow Transparent which are listed as immune in West Virginia are reported as resistant in most other states. From this it seems doubtful if there are any varieties which show a real immunity.

Control or Prevention.—The cedar rust of apples may be completely eliminated from an orchard by interrupting the life cycle of the pathogene by the removal and destruction of cedar trees. The preventive practices which relate to the alternate host, the cedar, are as follows: (1) Remove and destroy all cedar trees within a radius of 1 mile around the apple orchard. While this will not give absolute protection, it will afford commercial control in most cases. (2) Avoid the use of cedars for wind breaks around orchards or for ornamental plantings within a mile of apple orchards. (3) In case of a few small cedars in public parks or on private estates, the removal of the cedar apples previous to the production of the gelatinous sori will afford protection to nearby apple trees. For large trees this is a difficult and laborious task, and should not be attempted. (4) In cases where the cedar rust is threatening the life of the cedars and these are considered too valuable to destroy, some protection is possible by spraying the cedars. Spraying cedars with 6–6–48 Bordeaux plus 3 pounds of soap on July 26, and Aug. 6 and 15 gave 48 galls on the sprayed tree as compared to 950 on the control (Heald, 1908). The practical application of spraying cedars for the prevention of "cedar apples" must depend largely upon local conditions. The reduction in the number of galls is not sufficient to be of much value in preventing the infections of adjacent apple trees, but if the life of valuable cedars is being threatened by the abundance of the fungus, spraying should reduce its ravages sufficiently to prevent any material injury to the cedars.

The practices which relate to the apple are as follows: (1) Avoid planting apples, at least susceptible varieties like Wealthy, Rome and York, within the vicinity of established cedars. If the cedars cannot be eliminated or a safe distance cannot be found, select only known resistant varieties for planting. (2) If, as sometimes happens in the plains country, established cedars are prized more than a few apple trees, the removal of the latter will eliminate the disease on the cedars. (3) Spraying has been reasonably successful in the hands of careful experimenters, but it is unreliable as a commercial practice. Dusting has proved still more unreliable. Working in West Virginia, Giddings and Berg (1915) obtained the best control with lime sulphur, 1–40, slightly poorer with Bordeaux and still poorer with "atomic sulphur." Seven applications are necessary and these follow at short intervals: (1) when the blossoms buds are showing good color; (2) within 1 to 2 days after the first blossoms open; (3) when

one-half to two-thirds of the petals have dropped; (4) after 3 to 4 days; (5) after 5 to 6 days; (6) after 5 to 6 days; (7) after 6 to 7 days. Spraying, to be effective, must protect the foliage previous to the period of sporidial discharge, since it has been shown that infection requires only a few hours' time. The delay of a single day or part of a day may make the difference between failure and success. Since spraying must be so carefully timed and the different applications must be made at such short intervals, it would be difficult to secure effective control in large orchards without too great expense for labor and equipment.

In the regions in which cedar rust has become a real menace and the cedars are native and common, as in Virginia and West Virginia, there is no question but what the eradication of the cedar is the most effective and the most economical method of cedar-rust prevention. In these states special laws were enacted which declared the red cedar tree a nuisance and provided for its destruction. By this law a district could adopt "cedar local option." The cutting out of the cedars in Virginia and West Virginia has been practiced in many sections with excellent results and at an average cost of less than 50 cents per acre. "Cedar eradication is the cheapest form of orchard insurance you can buy. The cost on the average is less than the cost of a single spray application" (Fromme, 1918).

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IMPORTANT DISEASES DUE TO RUST FUNGI

ENDOPHYLLACEÆ

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Coleosporiaceæ

Several species of Coleosporium produce their pycnia and æcia on various species of pines, and their uredinia and telia on dicotyledonous hosts. The following is one of the most important.

Rust of golden rod and pine (*Coleosporium solidaginis* (Schw.) Thüm.).—Duggar, B. M.: *In* Fungous Diseases of Plants, pp. 435–437. Ginn and Company. 1909. Rankin, W. H.: *In* Manual of Tree Diseases, pp. 265–269. 1918. Hedgcock, G. G., Hunt, N. R. and Hahn, G. G.: New species and relationships in the genus Coleosporium. *Mycologia.* **12**: 182–198. 1920. —— and ——: Notes on some species of Coleosporium. I. *Mycologia,* **14**: 244–257. 1922; II. *Ibid.* **14**: 297–310. 1922. Weir, R.: The genus Coleosporium in the northwestern United States. *Mycologia* **17**: 225–239. 1925. Baxter, D. V.: A preliminary study of *Coleosporium solidaginis* (Schw.) Thüm. in forest plantations in the regions of the Lake states. *Papers Mich. Acad. Sci.* **15**: 245–258. 1930.

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Cronartiaceæ

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White-pine blister rust is a forest-tree disease of outstanding importance in the eastern United States and Europe and is now menacing the white-pine forests of the Pacific Northwest. Several other species of Cronartium of lesser importance are present in America:

	0, I hosts	II, III hosts
Eastern gall rust, *C. cerebrum* (Pk.) H. & L.	Pine (27 species)	Oak (29 species)
Western gall rust, *C. harknessii* Meinecke	Pine (13 species)	Castilleja, Orthocarpus and Pedicularis
Lodgepole pine blister rust, *C. filamentosum* (Pk.) H. & L.	Pine (3 species)	Castilleja, Orthocarpus and Pedicularis
Pine-Comandra rust, *C. pyriforme* (Pk.) H. & L	*P. ponderosa*	Comandra
Pine-Sweet gale rust, *C. comptoniæ* Arth.	Pine (17 species)	Comptonia and Myrica
Piñon blister rust, *C. occidentale* H. B. & H.	Pine (4 species)	Ribes

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MELAMPSORACEÆ

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Leaf rust of hemlock (*Necium farlowii* Arth.).—ARTHUR, J. C.: *N. Amer. Flora* **7**: 114. 1907.

Poplar rusts (*Melampsora spp.*).—Three different poplar rusts may be noted:

M. medusæ Thüm. Cæoma stage on larch.

M. abietis-canadensis (Farl.) Lud. Cæoma stage on hemlock.

M. albertensis Arth. Cæoma stage on fir. RANKIN, W. H.: *In* Manual of Tree Diseases, pp. 159; 182–183; 212–213; 298–300. The Macmillan Company. 1918.

Willow rust (*Melampsora bigelowii* Thüm.).—This rust produces its cæoma stage on the larch. There are several other species of Melampsora on willows. JACKSON, H. S.: The Uredinales of Oregon. *Brooklyn Bot. Gard. Mem.* **1**: 209–211. 1918.

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P. minimum (Schw.) Arth. Uredinia on *Rhododendron spp.*

P. myrtilli (Schum.) Arth. Uredinia and telia on *Vaccinium spp.* SPAULDING, P.: Diseases of the eastern hemlock. *Proc. Soc. Amer. Foresters* **9**: 245–256. 1914. HIRATSUKA, N.: Beiträge zu einer Monographie der Gattung Pucciniastrum. *Jour. Fac. Agr. Hokkaido Imp. Univ. Sapporo, Japan* **21**: 63–119. 1927. BOYCE, J. S.: A possible alternate stage of *Pucciniastrum myrtilli* (Schum.) Arth. *Phytopath.* **18**: 623–625. 1928.

Hydrangea rust (*Pucciniastrum hydrangeæ* (B. & C.) Arth.).—II and III on Hydrangea, the Peridermium stage on hemlock. ADAMS, J. F.: The alternate stage of *Pucciniastrum hydrangeæ*. *Mycologia* **12**: 33–35. 1920.

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Vaccinium rust or witches' broom (*Calyptospora columnaris* (A. & S.) Kühn.).—Affects species of blueberries and huckleberries. Peridermium stage on *Abies spp.* TUBEUF, K. F. VON AND SMITH, W. G.: *In* Diseases of Plants, pp. 370–373. Longmans, Green & Co. 1897. WEIR, J. R. AND HUBERT, E. E.: Notes on forest-tree rusts. *Phytopath.* **8**: 115–117. 1918. WEIR, J. R.: Observations on *Calyptospora columnaris* and *Peridermium ornamentale*. *Mycologia* **18**: 274–277. 1926.

PUCCINIACEÆ

Bean rust (*Uromyces appendiculatus* (Pers.) Fries).—An eu-auto-type. FROMME, F. D. AND WINGARD, G. A.: Bean rust. *Va. Agr. Exp. Sta. Bul.* **220**: 1–18. 1918. —— AND ——: Varietal susceptibility of beans to rust. *Jour. Agr. Res.* **21**: 385–404. 1921. WATERS, C. W.: The reactions of bean rust grown on leaves in solutions. *Papers Mich. Acad. Sci.* **5**: 163–177. 1926. ANDRUS, C. F.: The mechanism of sex in *Uromyces appendiculatus* and *U. vignæ*. *Jour. Agr. Res.* **42**: 559–587. 1931.

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Clover rusts (*Uromyces spp.*).—Three different clover rusts are recognized by Davis:

1. *U. trifolii-repentis* (Cast.) Liro on white clover (*T. repens* L.).
2. *U. trifolii* (Hedw.) Lev. on red clover (T. *pratense* L.) and zig-zag clover (*T. medium* L.).
3. *U. hybridum* Davis on alsike (*T. hybridum L.*).

All are eu-auto forms. KOBEL, F.: Trifolienbewohnende Rostpilze. *Mitteil. Naturf. Ges. Bern.* **1919**: 53–54. 1920. DAVIS, W. H.: Summary of investigations on clover rusts. *Mycologia* **16**: 203–219. 1924.

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G. juvenescens Kern on red and Rocky Mountain juniper.

G. kernianum Bethel on the Utah juniper.

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CHAPTER XXVI

DISEASES DUE TO THE PALISADE FUNGI AND ALLIES

BASIDIOMYCETES

This group includes the most important forms of the true basidium fungi or Basidiomycetes, represented by the familiar toadstools, mushrooms, shelf or bracket fungi, puff balls, earth stars, birds'-nest fungi and

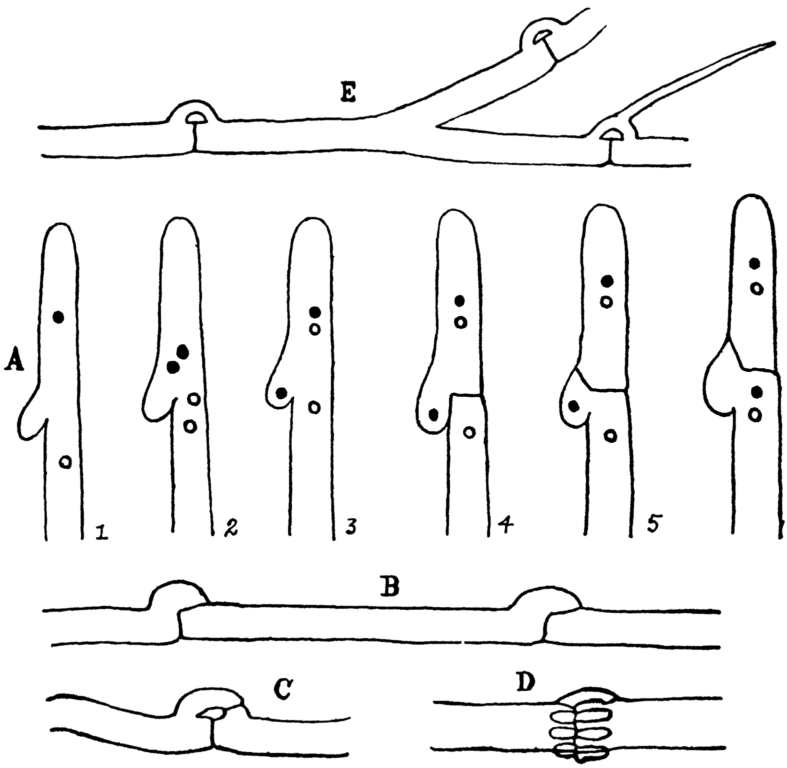

FIG. 230.—Clamp connections of basidiomycetes. *A*, the development of clamp connections. (*After Paravicini.*) *B*, *Stereum purpureum;* *C*, Rhizoctonia; *D*, a whorl of clamp connections in *Coniophora cerebella;* *E*, *Merulius lachrymans* with branch from one clamp connection. (*B–E, adapted from various sources.*)

stinkhorns. The familiar structures which are ordinarily referred to as "the fungus" are, in reality, the fruiting bodies only, while the mycelium or vegetative body of these fungi is hidden within the substratum, and must have made an extensive growth before the fruits or sporophores appear.

The Mycelium.—There are two rather distinctive features shown by the mycelium of most basidiomycetous fungi: (1) the binucleate condition of the cells of the hyphæ; and (2) the presence of characteristic forms of cell unions known as "clamp connections." While the binucleate condition of the cells is the general rule, uninucleate forms have been found, and multinucleate cells may be formed in the older tissues of the fruiting bodies by the amitotic fragmentation of the two original nuclei. The cells which give rise to the basidia are always binucleate. In fully developed clamp connections a slight bulging is noted on one side of the hypha just back of a cross-septum and this appears to overlap the beginning of the other adjacent cell. A clamp connection is formed by

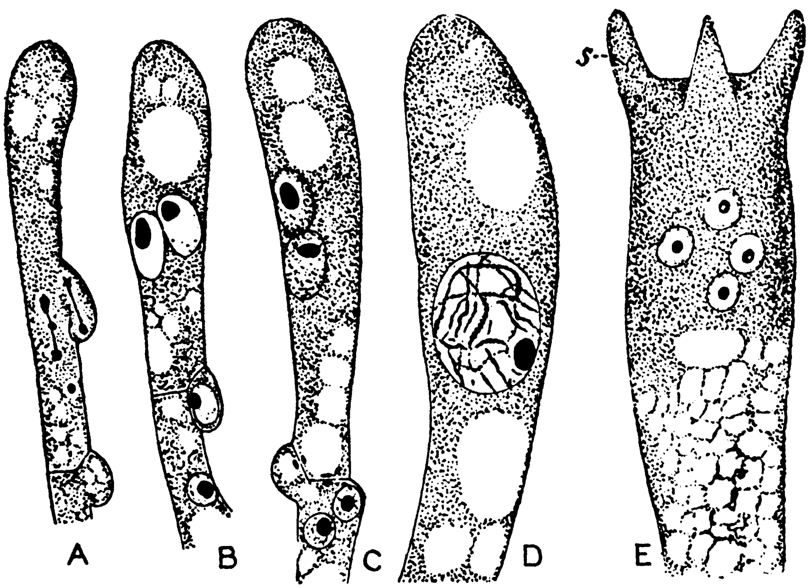

FIG. 231.—Development of the basidia of *Armillaria mucida*. (*After Kneip.*)

the growth of a short branch just back of a septum, which curves over until its tip comes in contact with the cell on the other side of the septum, when fusion takes place, bringing the cell contents of the two adjacent cells in communication. The opening is generally closed later, leaving the characteristic clamp. The presence of these connections is frequently a convenient means of recognizing a basidiomycetous mycelium. Rhizomorphs and mycelial plates are also characteristic features in some Basidiomycetes (see pp. 396–398).

Spores Types.—Conidiospores and chlamydospores are formed in some species, but these are inconspicuous and play a very minor part in the reproductive processes. The characteristic feature is the production of club-shaped hyphæ, the *basidia*, which form the continuous, uninucleate *basidiospores*, on slender terminal outgrowths, the *sterigmata.*

The common or typical number of basidiospores for each basidium is four, but in some species the number may be two, six or eight. The basidia are mostly arranged side by side in a more or less extensive palisade-like layer, the *hymenium*, which rests on a more compact subhymenial layer. Enlarged sterile cells, *cystidia*, may be mingled with the basidia.

In the simplest forms of Basidiomycetes, there is no differentiated or specialized fruiting body, the basidia being borne directly on an unorganized mycelial weft, but within the group there is great diversity in the form and size of the organized fruits, or sporophores. The progressive tendency from the simple to the more complex forms is the increase of surface over which the *hymenium* may be spread, with the display of the basidia in such a way as to secure wide dissemination of the spores. This increase of surface is attained in a great variety of ways, by wrinkles, folds, spines, teeth, pits, tubes or plates. The sporophore is thus simply an aggregate of supporting tissue, arranged in various ways for the efficient display of the basidia in large numbers.

Types of Sporophores.—In the true basidiomycetes three general types of sporophores may be recognized:

1. Beginning with the simple unorganized display of the basidia upon the mycelial weft, the sporophores may be first simple, prostrate or resupinate structures with a plain basidial surface, or these may be folded, rolled or shelving; next, increase of surface may be attained by the raising of the hymenium on the surface of cylindrical clubs, which may be very much branched or coral-like; or a third modification is seen in the formation of minute warts, short spines or flattened or cylindrical (simple or branched) teeth may rise vertically from resupinate sporophores or hang down from the lower surface of shelving fruits; a fourth type obtains the increased surface by shallow pits, honeycomb-like compartments or slender cylindrical tubes packed closely together; while the last device is the arrangement of thin plates or lamellæ packed closely together but not in contact. In all of these devices the fruit is open, that is, the hymenium is exposed so that the separated spores may be readily set free into the surrounding air. These fruits are characteristic of the Hymenomycetales or Agaricales, the true palisade fungi, which furnish numerous parasitic or semiparasitic species.

2. The second type of basidium fruit is a closed structure, in which the fertile or basidial portion, the *gleba*, is enclosed by a firm surrounding membrane, the *peridium*. The spores are set free only by the rupture of the peridium, with the formation of a definite opening, by irregular ruptures or by decay. These forms are illustrated by the common and the stalked puff balls, the earth stars, the hard-skinned puff balls, the sphere-throwing fungi and the birds'-nest fungi. These fruits are characteristic of the Gasteromycetales, an order containing only saprophytic species.

3. In the third type the gleba or basidial portion of the fruit is at first enclosed within a subterranean, tuber-like structure, which at maturity ruptures and allows the viscid spore mass to be carried rapidly upward by the expansion of an interior mass of elastic tissue. This type is illustrated by the common stinkhorn or carrion fungus, *Ithyphallus impudicus* and related forms, and is characteristic of the order Phallales. Species of this order are of but little interest to the plant pathologist, but it may be noted that *Ithyphallus impudicus* has been studied in connection with a disease of sugar cane.

Classification.—The importance of the Hymenomycetales or Agaricales in furnishing plant pathogenes and wood-destroying fungi will justify the characterization of the families and the most important genera:

1. Exobasidiaceæ, or the gall-forming palisade fungi. In this family, which consists entirely of obligate parasites, two features are characteristic: (*a*) an internal mycelium, which generally causes hypertrophy of leaves, fruits or other affected parts; and (*b*) the display of the *hymenium* over the surface of the galls or hypertrophied structures, rather than on organized sporophores.

Exobasidium.—Basidia four-spored and closely packed on the surface of the affected host parts. Parasitic on various species of the heath family (Ericaceæ).

2. Thelephoraceæ, or the smooth shelf fungi. Fruit a loose mycelial weft or a pellicular, fleshy membranous, tough fleshy or tough leathery structure, resupinate, rolled, shelving or branching, and with the basidial surface smooth on only slightly papillate or ridged. Basidia with two to six spores.

Septobasidium.—Sporophores coriaceous, resupinate and effused. Probasidia give rise to transversely septate basidia, bearing simple hyaline spores one each for the terminal and subterminal cells.

Corticium.—Sporophores hypochnoid, membranous, fleshy or leathery and always resupinate. Without cystidia. Spores globose to ellipsoid and hyaline.

Coniophora.—Sporophore membranous or fleshy membranous, resupinate and widely spreading, surface smooth or warty or wrinkled. Spores yellowish brown.

Stereum.—Sporophores leathery or nearly woody, resupinate or becoming shelving and frequently densely imbricated. Hymenium variously colored. Spores smooth, hyaline.

Thelephora.—Sporophores tough leathery, crustaceous, shelving-pileate or laciniately divided, and stalked or sessile. Hymenial surface generally covered with blunt warts, brown or grayish brown. Spores spiny, ellipsoid, often angular and brown.

3. Clavariaceæ, or the fairy clubs and coral fungi. Sporophores fleshy or tough, cylindrical or club shaped and unbranched, or branched simply

or in coralloid form, with cylindrical, flattened or even thalloid branches. Hymenium covering the clubs or branches. Basidia two- to four-spored, spores colorless or yellowish brown.

Typhula.—Sporophores filamentous, simple or slightly branched, hymenium covering the terminal slightly expanded or uniformly terete portions. Generally producing small seed-like sclerotia from which sporophores may develop.

Sparassis.—Sporophores fleshy and very much branched. Branches foliaceous, with hymenium on both surfaces. Spores hyaline and smooth.

4. **Hydnaceæ**, or the tooth fungi. Sporophores membranous felt-like, fleshy, corky, leathery or woody and resupinate, shelving or stipitate. Hymenial surface covering small warts, short spines, flattened or cylindrical teeth or slightly anastomosing tooth-like plates. In a few forms the pileus is lacking and the teeth spring directly from the substratum.

Hydnum.—Sporophores of varying consistency as indicated for the family and resupinate, shelving or cap-like. Hymenium covering terete or awl-shaped teeth which may be simple or branched in coralloid form. Spores colorless, smooth.

Echinodontium.—Sporophores shelving, woody, perennial, with closely set grayish-brown teeth on the lower surface. Interior of pileus and teeth reddish brown.

Steccherinum.—Sporophores pileate, sulcate and radiately subrugose. Hymenium on wide irregular teeth.

5. **Polyporaceæ.**—Sporophores annual or perennial, fleshy, leathery, corky or woody, and resupinate, shelving, or centrally or eccentrically stipitate, varying from small to very large. Hymenium covering the inner face of shallow pits, anastomosing wrinkles, or labyrinthiform lamellæ, but for the most part in slender, closely packed, cylindrical or angular tubes. Basidia four-spored, spores hyaline or colored.

Merulius.—Sporophores soft, almost gelatinous, soft cottony, or leathery and resupinate or somewhat raised or shelving. Hymenium covering vein-like ridges which anastomose in the middle to form shallow pits. Spores hyaline or brown.

Poria.—Sporophores membranous, fleshy, leathery or woody and completely resupinate, sometimes made up of only mycelium and tubes. Hymenium in short or elongated cylindrical or angular tubes, which generally are closely packed, but are sometimes scattered. Pores white or variously colored.

Fomes.—Sporophores woody, bracket-like or hoof-shaped, sessile or stalked, often concentrically zonate. Perennial and the closely packed tubes stratiform or superposed in annual layers, with the tissue between the tubes different from that in the general pileus.

Polyporus.—Sporophores at first fleshy but later becoming leathery, corky or woody, shelving or stipitate. Tubes developing from base

towards margin, similar to Fomes but never stratiform as the sporophores are annual. Spore powder white.

Polystictus.—Sporophores membranous or leathery, never woody, resupinate somewhat raised or distinctly bracket form. Annual, tubes never stratiform, developing from the center towards the margins.

Trametes.—Sporophores similar to Fomes and Polyporus, but with the pileus tissue extending between the tubes. Annual or rarely perennial but persistent.

Lenzites.—Sporophores shelving, sessile or narrowed at the base, leathery to corky-woody, generally tomentose and zonate. Hymenium of radiating gill-like plates with variable transverse connections, more pore-like towards the margin. Spores smooth, hyaline.

6. Agaricaceæ.—Sporophores generally soft fleshy but sometimes waxy or leathery, or typical mushroom form with cap or pileus and central or more rarely excentric stipe, or sessile and shelving. Hymenium covering radiating plates or lamellæ, the "gills," which are generally separate but may anastomose somewhat on the stipe. Spore powder white or variously colored.

Marasmius.—Sporophores fleshy to leathery, tough and drying without decaying, but reviving when moistened, centrally stipitate or more rarely either excentrically stipitate or sessile. Stipe cartilaginous or horny.

Lentinus.—Pileus fleshy, leathery or corky, generally funnel-shaped. Stipe when present central, excentric or lateral, confluent with the pileus. Gills toothed on the margin.

Schizophyllum.—Sporophores leathery or almost woody, gray-hairy above, sessile. Lamellæ of varying lengths, splitting and recurving or rolling up at maturity, when dry. Sporophores are persistent xerophytic in habit and revive under moist conditions.

Pholiota.—Pileus fleshy, symmetrical, gills adnate, veil forming an annulus. Spores brown to rusty.

Armillaria.—Sporophores fleshy, of typical mushroom form, gills at first covered by a veil which leaves a distinct ring or annulus on the stipe. Gills usually decurrent, whitish or becoming yellowish. Spores globular or ovate, hyaline.

Pleurotus.—Sporophores fleshy, laterally sessile or excentrically stipitate.

Palisade Fungi as Agents of Wood Disintegration.—The majority of the palisade fungi of economic importance are wood-destroying organisms, but a few are able to grow on herbaceous substrata. There are three agencies responsible for the destruction of timber while still standing in the forest or after it has been worked into various products. These are fire, insects and wood-destroying fungi, the last working mostly as silent or hidden enemies. The wood-destroying fungi may be purely

saprophytic, growing only on dead or structural timber, but in many cases these are of as great concern as definite parasites, since they may bring about the disintegration of foundation timbers, posts, poles or any timber which is in contact with moisture. The various wood-destroying fungi which invade living trees are for the most part *wound parasites*, gaining an entrance through pruning wounds, sun-scald cankers, winter-injured branches, mechanical injuries from hail, lightning or wind, insect tunnels, basal fire burns or various other injuries which may expose the wood to attack.

Certain species of palisade fungi confine their attacks very largely to the roots or basal part of the trunk, and thus bring about their disintegration, or cause a *root rot*. Other species make their best development in the heartwood, or in the portion of the tree trunk containing only dead tissue, and are unable to advance into the outer or sapwood. These *heart rots* may so permeate the heartwood as greatly to weaken the mechanical support which it should furnish, and also render the timber of little value for structural purposes. Some species which start in the heartwood may advance into the living sapwood and bark and cause the death or disintegration of the living cells. Other species with more of a parasitic tendency may be able to establish themselves at once in living sapwood or bark, without first growing for a time in dead tissue, thus bringing about disintegration which may be called a *sap rot*.

Mature sporophores produce spores in countless millions. One can be convinced of the enormous number by placing the pileus of a mushroom form gill face down upon a sheet of paper covered with a bell glass, when in 12 to 24 hours a heavy deposit of spores will fall making a "spore print" marking the position of the spaces between gills. In nature these spores are forcibly ejected from the basidia and are constantly falling when conditions are favorable for sporophore activity. These invisible clouds of spores are carried away by air currents and, if brought into open wounds of suitable hosts or woody substrata, may germinate and establish a mycelium.

The various wood-destroying fungi, by processes connected with their nutrition, cause the disintegration of wood. They must obtain much of their food from cell-wall substance, and the foods which they consume must be digested or brought into soluble or dialyzable form. Woody tissue has undergone a special modification known as lignification, that is, the cell walls contain lignin in addition to cellulose, while the middle lamellæ have a still different chemical composition. The solution of the different constituents by the invading fungi is brought about by enzymes or digestive ferments secreted, of which many different kinds have been isolated. Three of the marked changes which result may be noted: (1) the digestion of the lignin, leaving the cellulose; (2) the digestion of the cellulose of the walls, leaving the lignin; or (3) the digestion of the

middle lamellæ, thus causing a separation of the individual vessels. Invaded wood may become discolored, brittle, soft or punky in accordance with the character of the changes induced by the intruding fungus Complete disintegration may occur at localized points, leaving pockets which are sometimes filled with white aggregates of mycelium.

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THE RHIZOCTONIA DISEASE OF POTATOES

Corticium vagum B. & C.

This is a widespread disease of the Irish potato characterized by the production of stem lesions below the ground level, the appearance on the tubers of black specks in the form of "dirt that will not wash off," various minor injuries and secondary or accompanying effects of rather uncertain relationship. Various names, such as "black speck," "black-speck scab," "black scab," "scurf," "black scurf," "russet scab," "Rhizoctonia rot" and "little potatoes," have been applied because of the tuber characters

of affected plants, while "brown stem," "stem rot," "stem canker" and "potato collar fungus" are names based on the nature of the stem attacks. Some other names suggested by secondary or accompanying symptoms have been used, such as "rosette," "leaf roll" and aerial potato."

History and Geographic Distribution.—The early history of Rhizoctonia diseases is somewhat confused by the failure of writers to distinguish the two common species which are now recognized as *Rhizoctonia solani* Kühn and *R. crocorum* (Pers.) DC. The latter species has been described in recent years under the name of *R. medicaginis* DC. or *R. violacea* Tul. and has been given more attention as a parasite of alfalfa, beets, carrots, etc., than as a potato pathogene. The common Rhizoctonia of the potato was first recognized by Kühn (1858), who described the disease of the potato which it caused, and gave it the name of *R. solani*. This was recognized as a potato parasite by later European pathologists, and the disease of the potato which it caused was described as "Grind" by Sorauer and as "die Pockenkrankheit" by Frank in 1895. The first American report of a disease due to *Rhizoctonia solani* was by Pammel (1891), but he referred the beet-rot fungus which he studied to *R. betæ* Kühn. Atkinson (1892–1895) studied a "sterile" fungus as the cause of "sore shin" in cotton and the damping-off of other seedlings, but at the time did not recognize the form as Rhizoctonia. The first report of Rhizoctonia on potato in America was by Duggar and Stewart (1901). It was studied by Rolfs in Colorado (1902, 1904) and unusual importance attached to the disease in his two bulletins entitled "Potato Failures." He discovered the fruiting stage of the fungus, proved its relation to the subterranean mycelium and, based on the identification by Professor Burt, referred the parasite to *Corticium vagum* B. & C. var. *solani* Burt. This basidial stage had previously been recognized on potato stems by Prillieux and Delacroix (1891) in France and described as *Hypochnus solani*, although at that time the connection with the potato Rhizoctonia was not suspected. Selby published studies on the "rosette disease" of potatoes (1903) in which he stated that "the sterile fungus, Rhizoctonia, is indicated as the cause in the instances stated, by its constant presence," but in the light of present information his conclusions were too hasty.

There is a voluminous literature on Rhizoctonia as the cause of disease of other hosts than the potato, and since the pioneer work already cited various American and foreign writers have dealt with various phases of the Rhizoctonia problem. Special mention may be made of the demonstration by Gloyer of the ineffectiveness of formaldehyde for seed disinfection of Rhizoctonia-infected tubers (1913), the careful review of our knowledge concerning *R. solani* and *R. crocorum* by Duggar (1915), the general discussion of the "Parasitic Rhizoctonias of America by Peltier" (1916), the studies of Richards on the temperature relations of the disease (1921, 1923), and the recognition of physiological strains by Matsumoto (1921), Britton-Jones (1924) and Thomas (1925). Morse, Shapovalov, Drayton, Ramsey, Güssow, Edson and others have made valuable contributions to our knowledge of the disease and the more recent literature presents a mass of conflicting reports on the effectiveness of seed disinfection.

R. solani, on either the potato or some of its other hosts, is known throughout the United States and Canada, South America and the West Indies, India, Japan, Australia and New Zealand. It seems to be prevalent to a greater or less extent wherever potatoes are grown. Evidence points to its occurrence in raw desert lands of the Pacific Northwest (Pratt, 1918). Cases are on record in which the first crop of potatoes grown from clean treated seed and planted on virgin soil showed 50 per cent of the tubers heavily spotted with sclerotia. For a time it was thought to cause the most serious potato disease of many sections, especially the more northern regions, but this was due in part to the confusion of Rhizoctonia with virus disease symptoms.

It seems to reach its greatest severity in the northern states and Canada from Maine to the Pacific Northwest. The reduction in yield from Rhizoctonia for 1922 was estimated by the Plant Disease Survey, U. S. Department of Agriculture, to vary from traces to 8 per cent with one state reporting 15 per cent loss. According to current estimates it is much less serious than mosaic and leaf roll.

Symptoms and Effects.—The grower is most familiar with the Rhizoctonia disease as the "dirt that will not wash off" on the tubers, in the form

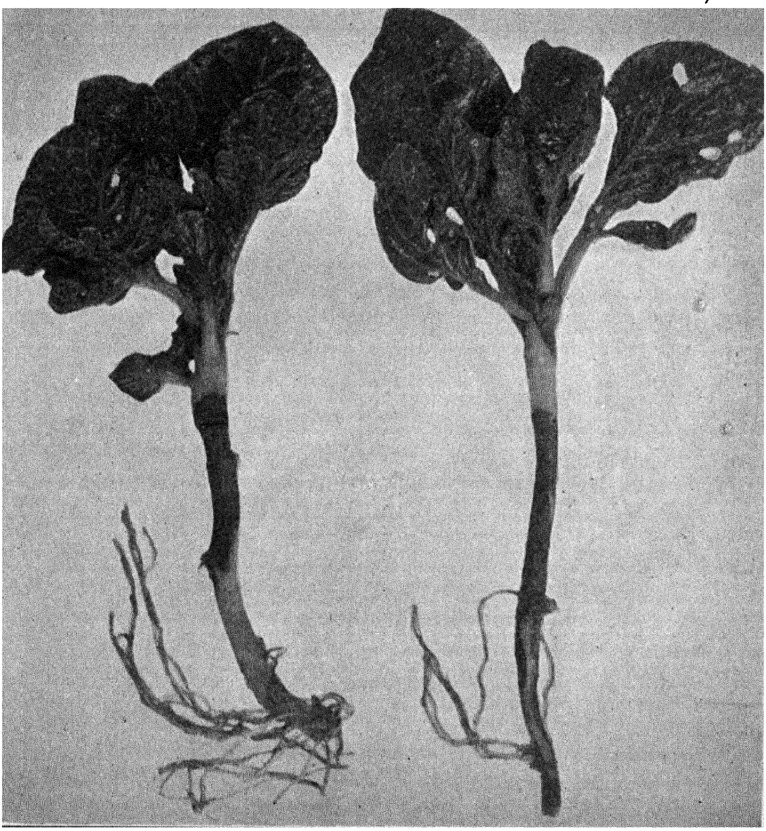

FIG. 232.—Young potato plants showing lesions due to Rhizoctonia.

of black particles of varying size. These black bodies or *sclerotia* are fungous structures which have been developed by the parasite during the growing period. If tubers are washed to remove all soil particles the sclerotia become much more conspicuous and appear of a deeper brown or black color. This is the stage that has suggested the names of "black scurf," "black scab," "black speck" or "black-speck scab." Under certain conditions smooth-skinned potatoes may show a roughened or checked condition designated as russet scab, which has been attributed to Rhizoctonia. A more severe and deeper scab somewhat resembling Actinomyces scab has also been attributed to Rhizoctonia, but most of

the evidence of the causal relation to this condition is based on association rather than experimental proof (McAlpine, 1911). While the rotting of tubers is not a common phase of the disease, such effects have been noted by various writers. This effect was described by Rolfs (1903) for seed tubers; McAlpine (1911) described a condition similar to late-blight rot which he thought was caused by Rhizoctonia; and more recently Shapovalov (1922) has reported Rhizoctonia as "an important causative agency

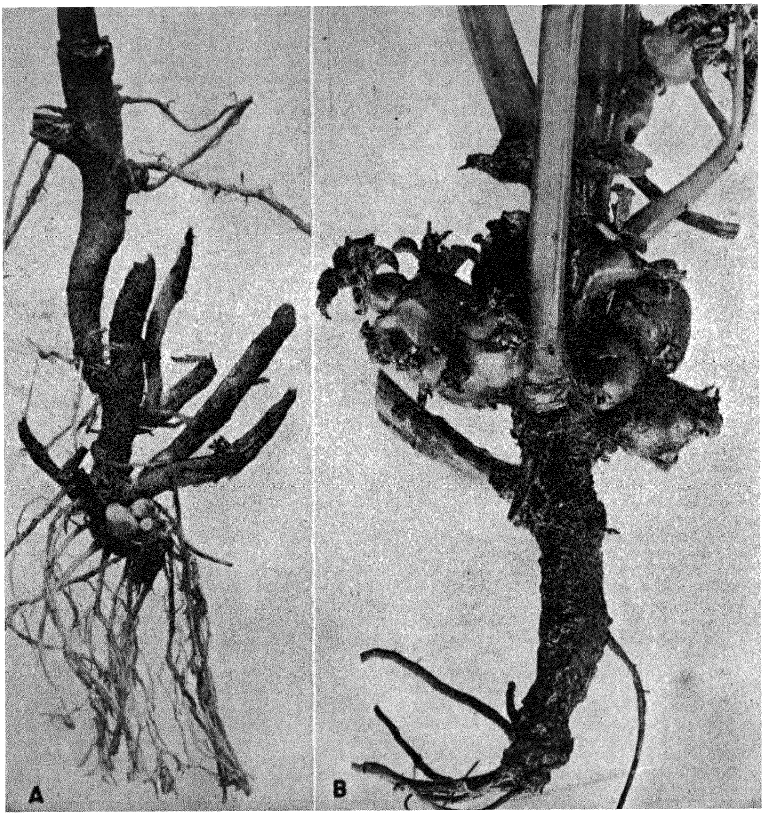

FIG. 233.—Effects of Rhizoctonia on potato plants. *A*, stems cut off by the fungus; *B*, stem lesion and aerial tubers. (*Photographs by B. F. Dana.*)

in bringing about the jelly type of decay of elongated stem ends of Burbanks and Netted Gems in the West." Whether Rhizoctonia unaided by other agencies can cause open pits by the corrosion of the tissue is uncertain (Morse and Shapovalov, 1914; Ramsey, 1917), but it probably does follow insect or other injuries and extend the pits or channels. There is little proof that the irregular and malformed tubers described by Morse and Shapovalov (1914) as an effect of the disease are really Rhizoctonia effects.

If young sprouts of affected plants are examined early in the growing season, the stems below the surface of the ground will frequently exhibit elongated, reddish-brown or very dark lesions which are in marked contrast to the whitish or slightly yellowish surfaces of the normal tissue. These lesions may extend for an inch or two along a stem on one side or the stems may be completely girdled. The amount of injury depends on the extent and penetration of the lesions. In some early attacks the first

FIG. 234.—Corticium stage on stems and leaves of potato.

young sprouts may be cut back or "burned off" by these lesions, and later crops of sprouts may suffer a like fate. In the extreme cases none of the sprouts are able to reach the surface, and missing hills result, but in many less severe attacks the later sprouts originating below a lesion, or directly from the seed piece, reach the surface and produce nearly normal tops. If the lesion is slow in developing or infection takes place later in the growth period, the affected stems may show basal corrosion and drying which

interfere with the normal life processes, although causing little reduction in size of the top. Lesions on stolons may interfere with the setting of tubers. The girdling of stems close to the surface of the soil may cause

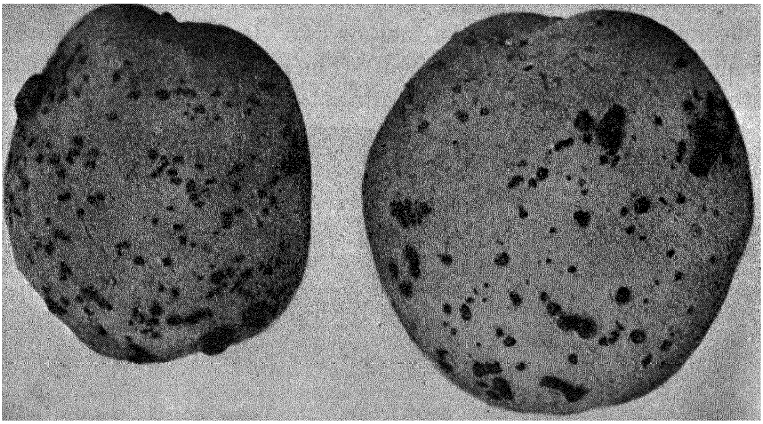

FIG. 235.—Potatoes showing numerous sclerotia of average size. (*Photograph by B. F. Dana.*)

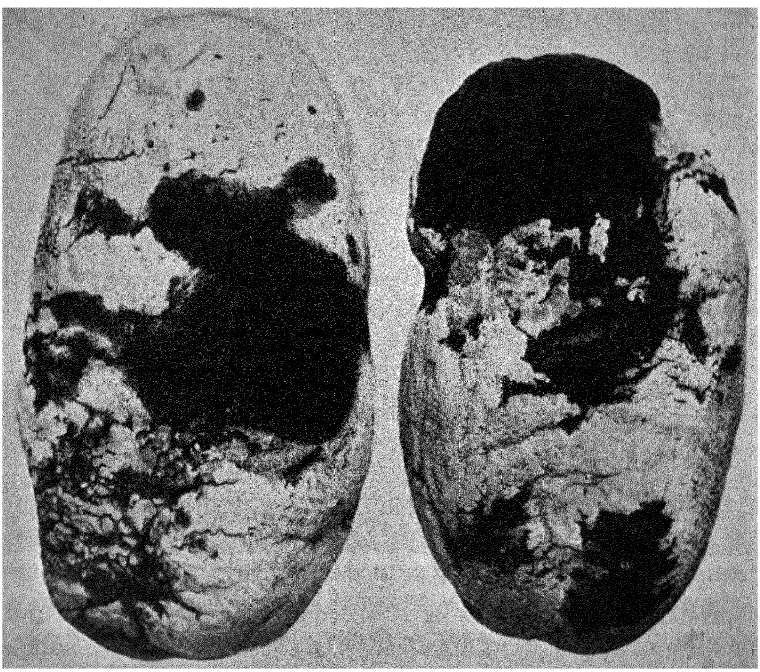

FIG. 236.—Potatoes showing giant sclerotia of Rhizoctonia. (*Photograph by B. F. Dana.*)

the production of aerial axillary tubers, but this is no diagnostic character for Rhizoctonia. Yellowing and rolling of leaves may also accompany severe Rhizoctonia attacks.

It is not uncommon for the vegetative body of the causal fungus to develop in such profusion on the stems and roots as to make a conspicuous brown mantle of interlacing threads which can be readily detected when the affected parts are removed from the soil. This external mycelium may sometimes be very abundant without causing any definite lesions on roots, stolons or stems, while in other cases lesions may be quite evident and the fungous filaments relatively inconspicuous.

From early July and during the rest of the growing season while the tops are still green, the stems just above the ground level may be covered with an evident white, powdery crust, which looks to the naked eye almost like a chemical incrustation. This may be faint or very conspicuous and extend up the stem for several inches. Its presence is a certain indicator that the vegetative body of the parasite is already well established on the underground organs. Its presence is no indicator, however, of the amount of damage from the disease. The author has seen this fruiting stage of the parasite in abundance, on a field of potatoes which gave a higher yield then the average fields of the vicinity.

The injury from Rhizoctonia is exceedingly variable and has undoubtedly been overemphasized by many writers. Tubers which show the sclerotia even in abundance are not really injured for table stock, but their value is lessened somewhat because of the disfiguring effect. Sclerotia-bearing tubers are less valuable for seed stock, since it is recognized that they may carry the disease into disease-free fields, even though seed disinfection is practiced. Reports as to the amount of actual damage to the crop are quite conflicting for different sections of the country, and vary from almost complete crop failure to no appreciable reduction in the yield of table stock. In estimates of the damage from the disease too much importance has been attached to the abundance of the sclerotia on the tubers and too little to the real effect on yield. The number of sclerotia-bearing tubers and the number of sclerotia will vary with the soil moisture and soil temperatures and generally increase from maturity to digging time. An increase from 7.5 per cent on Aug. 20 to 93.5 per cent on Oct. 13 has been recorded (Bisby *et al.*, 1923). Under conditions in eastern Washington the writer has never been able to increase the yield of table stock by seed disinfection, even though heavily contaminated seed was used in all cases. In other localities very beneficial results have been claimed. Under conditions in which severe stem and sprout injuries characterize the disease, the crop may be expected to suffer reduction in both quantity and quality.

The common Rhizoctonia disease of the potato is not to be confused with the violet Rhizoctonia (*R. crocorum* DC.), a trouble that has been noted on the potato in the Pacific Northwest and in Nebraska (Faris, 1921). In this disease the tubers show a very conspicuous mantle of

brownish-violet or violet mycelium and mycelial strands, ordinary sclerotia and numerous microsclerotia (infection cushions).

Etiology.—This disease is due to a simple basidiomycetous fungus, *Corticium vagum* B. & C., which in its sterile mycelial stage has been described under the name of *Rhizoctonia solani* Kühn. It has been clearly proved by pure culture inoculations that this parasite is able to form the characteristic stem or stolon lesions, and that it can induce a rotting of tubers. At times, however, the mycelium may be present in abundance on the surface of stems, roots and tubers, with little or no evidence of

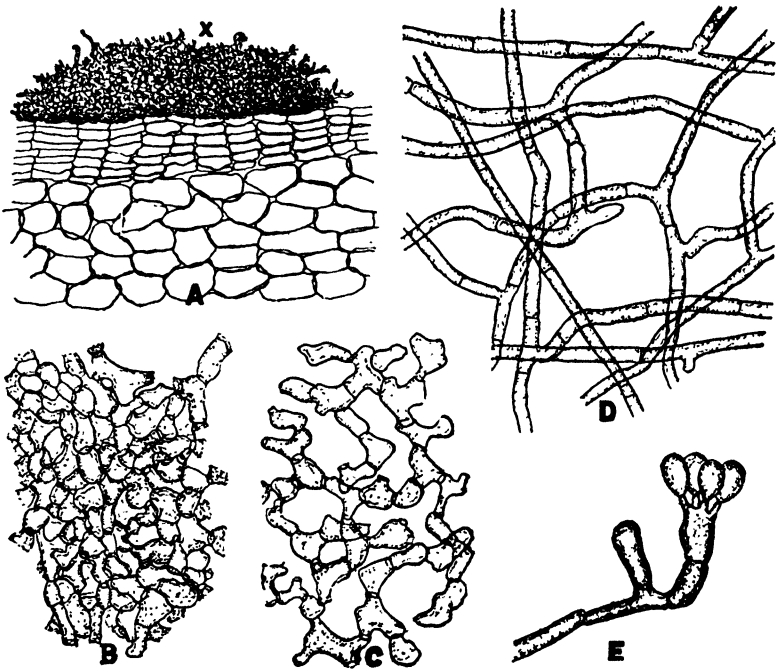

FIG. 237.—*A*, portion of potato skin showing section of a sclerotium; *B*, cells from most compact sclerotial tissue; *C*, cells from loose sclerotial tissue; *D*, distributive hyphæ from surface of a tuber; *E*, a basidium and basidiospores of *Corticium vagum*. (*After B. F. Dana.*)

disturbance in the life of the host. It has been shown, however (Edson and Shapovalov, 1918), that *R. solani* Kühn is not the sole cause of the familiar stem lesions, but that various other soil fungi, acting independently or in conjunction with Rhizoctonia strains, are also causally related. From the early work of Rolfs (1902, 1904), Selby (1903) and others it seems that the diversity of the Rhizoctonia effects on the potato has been somewhat exaggerated. The widespread occurrence of *R. solani* and the reputation which it gained as a parasite led to erroneous conclusions which were based largely on the presence of the parasite. In some sections it is almost impossible to find potatoes that are free from the fungus, and in certain cases the effects of virus diseases have thus been

attributed to Rhizoctonia on purely circumstantial evidence. Whether Rhizoctonia can act on the root system of the potato in the way suggested by Güssow (1917) seems somewhat doubtful. The author observed this killing of the absorbing roots, and the subsequent early death of the tops without the appearance of the characteristic stem lesions, and at first attributed the effects to Rhizoctonia, but later tests have shown that one of the virus diseases was the real cause, since all the symptoms were duplicated in carefully treated seed from hill selections in sterilized soil in which no trace of Rhizoctonia could be detected. It seems certain that leaf roll, mosaics, russet dwarf, witches' broom and other degeneration diseases have been confused with the Rhizoctonia disease. There appears also an element of doubt as to the causal relation of Rhizoctonia to the pits as described by Ramsey (1917) from Maine material. This condition has also been noted in the Pacific Northwest, and while Rhizoctonia has generally been found in the corroded tissue, the cysts described for potato pox have also been found.

From evidence now in hand it seems that many of the secondary or accompanying symptoms are not, in reality, to be attributed to the action of the parasite, but to either environmental factors or to virus diseases of the various types. Some of these are the "rosette disease" as described by Selby (1903), large tops but no tubers (Rolfs, 1902, 1904), little potatoes, aerial tubers, tubers but no top, irregular or branched tubers, deformed, split or cracked tubers, and grouping of tubers close around the stem. While Rhizoctonia may play a part in some of these effects, it is clearly a minor or secondary factor.

Rhizoctonia may be present on the subterranean portions of the potato as a superficial *mycelium* or in the form of mycelial aggregates, or *sclerotia*, while the sporulating or *basidial stage* develops upon the stem above the soil, on leaf petioles or leaflets close to the soil or sometimes on the surface of the soil. The young vegetative hyphæ are colorless, vacuolate, septate at intervals of 100 to 200μ and invariably show branches that are more or less constricted at their points of origin from the main axis. The hyphæ soon become colored and then are evident as a web or weft of yellowish-brown or brown strands, frequently so abundant as to be evident to the naked eye, but sometimes so few as to require microscopic examination for their detection. The most mature hyphæ are very dark, rather rigid, the cell walls thicker, the cells of uniform diameter (8 to 12μ) and the branches arise generally at right angles to the main axes. In the organization of sclerotia denser tufted groups of hyphæ are formed with profuse branching, shorter cells of irregular diameter, showing lobulated, elbowed or moniliform types. In the case of sclerotial formation this type of growth becomes still more compact.

The sclerotia are produced most abundantly on the tubers and vary from minute cell groups barely visible to the naked eye to giant sclerotia

an inch or more in diameter. They are rounded or irregular, more or less flattened, dark brown or almost black, smooth on the surface and are quite easily detached from the skin, which is not generally penetrated by any of the hyphæ. Drayton (1915) has recorded the occurrence of sclerotia within the cortical tissues of stem lesions and the penetration of the hyphæ into the vascular bundles and pith cells. The most compact sclerotia appear in sections to be made up of closely grouped cells with few intercellular spaces, a pseudoparenchyma, but less dense sclerotia may show a spongy structure of short irregular "bloated" cells with constrictions at the cross-septa.

Under suitable conditions of moisture the network of brown hyphæ which clothe the stem advance upwards around its base and form a felt-like mantle of white fruiting hyphæ. Numerous branches produce typical basidia upon the surface of the mantle and a white, powdery appearance is characteristic of the mature sporulating condition. Each basidium produces four sterigmata, each of which produces a hyaline, elliptical or obovate basidiospore, 9 to 15 by 6 to 13μ. This fruiting stage was described as *Hypochnus solani* by Prillieux and Delacroix (1891) and this name has been generally retained by European writers, who believe the fungus should be referred to the Hypocnaceæ. American usage is based on the determination of Burt from the study of the potato collar fungus studied by Rolfs in Colorado (1903).

The abundance of the basidial stage is probably accountable for the widespread occurrence of the fungus. Potatoes grown from heavily infected seed have been noted with 70 per cent of all the hills showing the sporulating stem collars. The basidiospores are forcibly abjointed from the sterigmata and are wind-disseminated. They are able to germinate at once and establish new mycelia either in the soil or in connection with other hosts. The parasite is also spread very generally by the use of potato seed stock that is carrying numerous sclerotia. Under conditions favorable for the growth of the potato the overwintered sclerotia soon develop an active mycelium by the production of hyphæ from many of their component cells. When an infected potato crop is harvested, sclerotia and also vegetative mycelia are left behind in the soil and are ready to attack any susceptible crops that may follow on the same land.

Predisposing Factors.—The most important factors influencing the severity of the Rhizoctonia disease of potatoes are the soil reaction, the fertility of the soil and the soil temperatures which prevail, especially during the early part of the growing period.

R. solani makes its best growth on an acid or slightly alkaline medium and consequently seems to thrive best in acid soils. Its behavior is just the opposite of the common scab organisms, which develop best in alkaline soils. Liming the soil for the correction of acidity has been reported to afford some relief, but it has not been uniformly successful. Fertilizers

that increase the acidity of the soil—for example, acid phosphate—will make conditions more favorable for Rhizoctonia and have been reported to increase the per cent of infected tubers. In some cases the application of sulphur has increased the severity of Rhizoctonia, while in others no appreciable effect has been noted. Liberal applications of barnyard manure have generally given a reduction in the per cent of infected tubers. The prevalence of favorable soil temperatures is the most important factor influencing Rhizoctonia infection and injury. Richards (1921) has shown that:

> The greatest damage occurred between 15 and 21°C., while 18°C. proved to be the most favorable temperature for tissue destruction as well as for growing-point injury. Serious destruction of tissue resulted at 9°C. The severity of attack decreased rapidly above 21°C. until at 24° *C. vagum* proved to be of minor parasitic importance. Few typical lesions occurred at or above 27°C.

This temperature relation will explain many of the conflicting results that have been obtained by different workers and will also explain why an early-planted crop generally suffers more from the disease than later plantings. The reason for greater injury to the early crop is apparently, then, based on the cooler soil temperatures, which favor the growth of the parasite during the early growth of the crop. These conclusions have been substantiated by both greenhouse cultures and by field tests. In practical application of these facts it may be noted that the temperatures which are most conducive to the Rhizoctonia injury are also the most favorable for the growth of the potato plant.

Host Relations.—An enumeration of all the hosts of *R. solani* or its strains would be beyond the scope of this treatment. In addition to the potato, many other cultivated plants are susceptible and many of the common weeds of our fields and gardens are known to harbor the parasite. At least four general groups of symptoms may be recognized: (1) the damping-off of seedlings or of cuttings; (2) the stem rots or root rots, or the rotting of root crops; (3) the rotting or blighting of foliage; (4) the rotting of fruits. *R. solani* ranks with *Pythium debaryanum* as an important agent of damping-off, and is probably more serious in this connection than as the cause of disease in older plants. The majority of the seedling hosts in which damping-off by Rhizoctonia has been studied belong to the Dicots, but some Monocts are known to be susceptible—the onion, for example—while coniferous seedlings frequently suffer severely. The damping-off by Rhizoctonia is not confined to seed beds, but is serious in both garden and field, as may be illustrated by the damping-off of beets and the sore shin of cotton. Damping-off of cuttings by Rhizoctonia in the propagating house is a phenomenon of frequent occurrence.

Herbaceous stems or roots of various hosts may be affected in much the same way as the potato, while woody stems may sometimes be

invaded. Beans, beets, carrots, carnation, eggplants, peas, radish and sweet potato are some of the more important adult herbaceous hosts. The author has found Rhizoctonia as the sole fungus in lesions on raspberry canes and on young pear trees. In fleshy roots like the beet, carrot or radish a crown rot has been noted in which the infection takes place at the bases of the leaves.

The invasion of the foliage is not so frequent as the other phases of the disease. One illustration may be cited in the case of lettuce, in which the leaves of mature plants grown under glass were rotted, the inner and younger leaves completely, the outer or older leaves all except the midrib. In such a case the primary invasion was through leaves in contact with the soil.

A number of cases have been studied of rotting of fruits due to Rhizoctonia. In beans grown on contaminated soil many pods in contact with the soil may be invaded during periods of moist or rainy weather. A rot of either green or ripe tomatoes is not uncommon and has been observed in numerous localities. The author has found it under even very dry conditions in Washington, and in such cases the Corticium stage has been developed on the side of the fruit in contact with the soil. The fruits of eggplants are sometimes severely rotted by Rhizoctonia, but these fruit attacks do not seem to be especially common. The author has also studied a rot of strawberries which was shown to be due to Rhizoctonia. A portion of a field showing foliage symptoms of the disease and numerous cases of sporulation on the leaf petioles yielded numerous berries in contact with the soil that were rotted by Rhizoctonia as proved by cultures. These also showed the Corticium stage of the fungus on the surface.

Biological Strains.—The existence of various physiologic strains of *R. solani* has been recognized, although their separation has not been so clearly defined as in numerous other pathogenes. Matsumoto (1921) recognized six strains from a study of 15 isolations from potato, bean, lettuce, dahlia, eggplant and Habernaria from California and Missouri, on the basis of cultural and physiological characters and pathogenicity. Britton-Jones (1924) studied isolations from cotton from Egypt, England, India and United States but concluded that all were strains of *R. solani*. Thomas (1925) studied strains from Europe, America and Java, showing variation in cultural characters, temperature requirements and parasitism. Low-temperature and high-temperature strains were recognized. *Moniliopsis aderholdii* was classed as one of the strains of *R. solani*, but this is contrary to the conclusions of Wellensiek (1925).

Prevention or Control.—No single practice will meet the needs of the potato grower, but several lines of procedure are available, and emphasis on these will probably vary in different environments. Seed selection, seed disinfection and cultural practices intended to avoid or lessen infection are all of value.

1. *Seed Selection.*—The use of sclerotia-free seed stock lessens the chances of heavy infection and prevents the introduction of the parasite in large quantities into new fields. In some cases selection alone has given better results than seed disinfection, but in many environments seed disinfection is necessary because of other troubles.

2. *Seed Disinfection.*—The common occurrence of seed-borne potato parasites, which can be killed by the application of a fungicide, has prompted a general recommendation of seed disinfection (for details, see Potato Scab, p. 375). For Rhizoctonia, which is more resistant than scab or blackleg, the following have been recommended: the standard cold corrosive sublimate, the modified acid-containing mercuric chloride (Leach *et al.*, 1929), the hot corrosive sublimate, and the hot formaldehyde. Organic mercury dusts and dips have given control under some conditions. As a result of the experimental tests of the last few years the whole matter of seed disinfection for Rhizoctonia control is in a rather unsettled condition, and the course to follow must be gleaned from a mass of contrary experiences. It now seems doubtful if seed disinfection is as valuable as formerly thought to be, since in many cases neither increased protection nor increased yields have resulted. Clayton (1929) and Gratz (1930) state that seed disinfection for Rhizoctonia control is not sufficiently profitable to justify the expense.

3. *Cultural Practices.*—Attention may be given to the following: (*a*) rotation to avoid contaminated soils, using non-susceptible crops, such as small grains or corn; (*b*) the avoidance of acid fertilizers, and the use of lime or manure, unless the prevalence of scab opposes it; (*c*) planting late enough to avoid the period of low temperatures especially favorable to the disease; and (*d*) harvesting as soon as mature to prevent the increase in the number of sclerotia by the tubers remaining in the ground when moisture and temperature conditions are favorable for the continued growth of the parasite.

Selected, sclerotia-free seed treated by one of the standard methods and planted with due consideration to cultural control may be expected to give the highest percentage of disease-free tubers that can be produced under the conditions which prevail.

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THE MUSHROOM ROOT ROT

Armillaria mellea (Vahl.) Sacc.

The mushroom root rot is a disease which affects various coniferous trees, many broad-leaved forest trees, various fruit trees, the grape, the bush fruits and various wild or cultivated shrubs, causing a rotting of the bark and wood of the roots and crown followed by death of the host. It has been designated as the shoestring-fungus rot, Armillaria root rot, crown rot, rhizomorphic root rot, toadstool disease, and on coniferous

trees the name "resin flow" or "resin glut" is sometimes applied. The causal fungus is generally referred to as the "honey agaric," the "oak fungus" or the "shoestring fungus." The German names are "Honigschwamm," "Honigpilz" and "Hallimasch"; the French, "pourridie" and "maladie des racines."

History and Geographic Distribution.—Although the causal fungus was described in 1777 as *Agaricus melleus* by Vahl, it was not until many years later that it was recognized as a parasitic form. Much of our early knowledge of this disease comes from the investigations of Robert Hartig (1874, 1878), who has been called the father of forest pathology. The first studies showed the disease to be very important in the forests of Germany on the various coniferous trees, and Hartig claimed that *A. mellea* was also parasitic upon *Prunus avium* and *P. domestica*, but saprophytic on other trees and contributed to the disintegration of the timber from various species. Hartig first showed the genetic connection between the rhizomorphs which had been known as *Rhizomorpha subterranea* Pers. and *R. subcorticalis* Pers. and the sporophores of *Agaricus melleus*. Previous to his work these peculiar structures had been believed to belong to entirely separate and distinct species of fungi. While De Bary, Brefeld and others contributed to our knowledge of this disease, the work of Hartig is of the most outstanding importance. Importance was attached to *A. mellea* as a cause of root rot of the grape by Dufour (1886) and by Schnetzler (1886), and according to Scribner (1890) was first discovered on this host in America in 1887 by Viala, who found it in Missouri, Texas and California. The mushroom root rot assumed importance as a prune disease in the Pacific Northwest and was studied by Piper and Fletcher (1903). Later work by Lawrence (1910) showed that it was serious on various fruit trees and on blackberries and raspberries, especially in the Puget Sound country. It was given special consideration as a disease of citrus trees in California by Horne (1912 and later) and of various hosts in Oregon by Barss (1913). Previous to this time a very similar trouble, now believed by some to be identical, was studied by Wilcox (1901) in Oklahoma and adjacent territory under the name of a "Rhizomorphic root rot of fruit trees," but it was attributed to *Clitocybe parasitica* n. sp. The prevalence of the disease in the central Mississippi had led von Schrenk and Hedgcock to a study of the trouble at about the same time. It is of interest to note that Armillaria root rot was studied in Australia in 1910 (Johnson) and in recent times has been given attention from the standpoint of control (Birmingham and Stokes, 1921). The disease continued to attract the attention of plant pathologists in European countries, mainly, however, as a forest-tree disease. Important contributions have been made by Cieslar (1896), Bolle (1913), Hey (1914), Voglino (1917) and Falck and Blatz (1918). While main consideration of the disease in the United States has been focused on its attack on fruit trees, Long (1914) has studied it as the cause of death of chestnuts and oaks. One of the most recent studies was made possible by Hiley (1919) through a special grant from the British Board of Agriculture and Fisheries for the study of the larch canker, Dasychypha.

Armillaria mellea was reported as causing a field rot of potatoes in Australia (1910), and was noted on the same host in Washington in 1912 (Bailey, 1914). It has since been reported on the potato in other western localities, also in Michigan and other eastern states.

From the above consideration it will be seen that Armillaria root rot is a disease of considerable importance in European countries, Australia, Japan and the United States. It is not mentioned in a recent list of South African diseases. In the United States it has attracted the most attention west of the Rocky Mountains in California, Oregon and Washington, and in the central Mississippi Valley, although it is not unknown in other sections.

Symptoms and Effects.—The first external symptoms of the disease are the decline in vigor and retarded growth of the plant as a whole or of certain branches. Leaves may remain undersize and scanty, turn yellow and fall prematurely, and branches may die back. A debilitated condition of this sort is not diagnostic for Armillaria root rot, but may follow other fungous troubles (*e.g.*, silver leaf (*Stereum purpureum*), bacterial collar rot (*B. amylovorus*) or collar rot due to winter injury). In coniferous trees there may be an abnormal exudation of resin (resinosis)

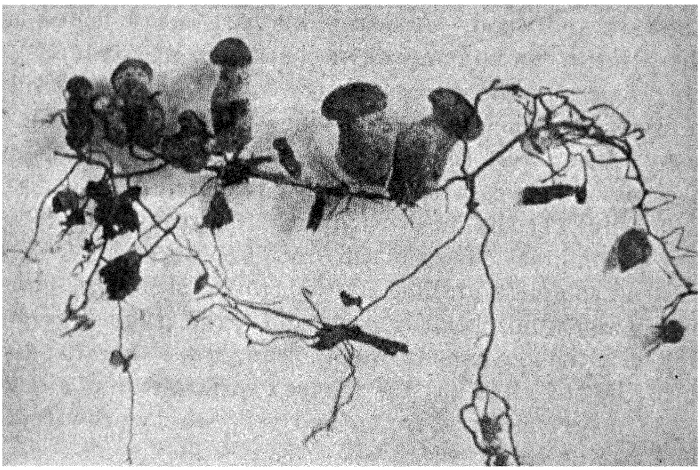

FIG. 238.—Rhizomorphs and young sporophores of *Armillaria mellea*. (*Photograph by H. H. Whetzel.*)

from the base of the tree, and "sometimes the quantity of this substance excreted is so great that the fallen needles, twigs and soil around the base of the trunk become compacted into a hard adherent crust" (Hiley, 1919). This resinosis is sometimes the first external symptom in coniferous trees, but browning and dropping on the needle leaves may be expected to follow. In stone and citrus fruits more or less gummosis may accompany the attack.

Trees or other hosts showing the symptoms outlined should be examined for diagnostic indicators. The soil should be dug away from the crown and large roots and a search made for: (1) areas of decayed bark or wood; (2) somewhat fan-shaped, felt-like growths of white mycelium which spread out between the wood and bark or within the bark in decayed parts; (3) shiny, dark-brown or black, root-like or cord-like strands, the *rhizomorphs*, $1/25$ to $1/12$ inch in diameter, which run over the surface of the crown or roots in a branched or anastomosing system, ramify between the bark and wood of old lesions or extend through the soil for some distance from the affected parts. While the mycelial character is quite distinctive, the detection of the rhizomorphs is unmistak-

able evidence of the true nature of the disease (Fig. 115). The later appearance of the characteristic groups of honey-colored toadstools around the crown of the affected plants will give final proof of the presence of mushroom root rot. The fruiting bodies do not always appear, and are generally not in evidence until the host is dead or in the last stages of decline, hence main reliance in diagnosing the disease can be placed on the presence or absence of rhizomorphs.

The injury from mushroom root rot will vary somewhat with the location of the zone of attack, and the rapidity of advance of the disintegration of bark and wood. A susceptible host once infected is doomed unless the parasite can be removed or checked in its advance. On some hosts the progress of the disease is very rapid, and fatal results follow the same season or the season following the initial infection, while in other cases (older forest trees) the tree may make a hopeless struggle through a period of years in a crippled condition. The disease may cause injury by: (1) the localized attacks on roots, followed by their death and the indirect effects of this root loss on aerial structures; (2) the partial or complete girdling at the crown, causing a physiological separation of remaining healthy roots and aerial parts or very seriously interfering with the movements of both crude and elaborated sap, ending in death of the tree; and (3) the reduced surface extent of the foliage, stunted and poorly matured fruits, and very scanty growth during the progress of the disease.

In orchards or in pure stands of timber the affected trees generally appear in spots or groups, and these if watched will be seen to increase in size as more trees become affected, the disease spreading from the original center of infection. Where the disease is of long standing and many foci exist, the affected areas may merge more or less, thus obscuring the points of origin. In Oklahoma the distribution of the "rhizomorphic root rot of fruit trees" followed the location of the timber belts (Wilcox, 1901). In Europe essentially the same relation has been noted in successions of forest trees, the infection becoming epiphytotic, especially when conifers follow broad-leaved trees. According to Hiley (1919), more trees die in Europe from the attacks of Armillaria than from any other parasite. In almond and citrus orchards in California it is noted that the centers of infection frequently coincide with places where oak trees formerly stood, the fungus spreading to the fruit trees from the remains of the old oak stumps or roots. Some idea of the amount of damage in prune orchards may be gained from the progress of the disease through a period of years, as shown by the tabulation on page 845 (Piper and Fletcher, 1903).

The explanation for this heavy loss is the fact that the prune orchards were planted on cleared land on which the native growth had been affected by the root rot.

Orchard No. 1: 1053 trees		Orchard No. 2: 5000 trees	
Year	Number dead trees	Year	Number dead trees
1895	6	1898	Disease evident
1896	77	1900	300
1897	40	1901	300
1898	64	1902	250
1899	154		
1900	125		
1901	117		
Total	583	Total	850

Etiology.—The mushroom root rot is due to *Armillaria mellea* (Vahl.) Sacc., a common and widely distributed gill fungus, which produces sporophores of the Agaricus or toadstool type. This fungus is able to lead a saprophytic existence on stumps and roots of dead trees, but under favorable conditions it becomes a serious wound parasite. Some observations and experiments have led to the conclusion that this pathogene cannot enter the normal unbroken tissues of roots or crown of some hosts, the healthy bark serving as an effective barrier, while there is evidence that in the case of potatoes, roots of citrus, etc., the rhizomorphs may penetrate directly into healthy tissue. It has, however, been shown that wounds are readily penetrated and also that dead roots are easily entered by the rhizomorphs. This peculiarity of the fungus will explain the increased severity of the disease when trees are suffering from unfavorable environmental factors which lead to localized root killing. Hiley (1919) offers this absence of dead roots as the explanation for the fact that larch woods remain practically free from infection until they reach an age of more than 15 years.

This fungus has no conidial stage, and relies entirely on the *rhizomorphs*, and the *basidiospores* produced in enormous numbers by the sporophores, for its dissemination. Two types of rhizomorphs are formed, *subcortical* and *free* or superficial. The former replaces the felted mycelium between the bark and wood, when the tree is dead and the bark becomes loosened, and consists of flattened whitish strands, which become colored when the separation of the bark exposes them to the air. They branch more profusely than the free rhizomorphs and some branches enter the host tissue, especially through the medullary rays. The free or subterranean strands or rhizomorphs are rounded instead of flat and consist of a brown cortex of closely compacted fungous tissue enclosing a central medulla of hyaline hyphæ arranged in longitudinal rows. The relative amount of cortex and medulla is variable, the former becoming thicker

with age. These rhizomorphs have an apical growing region by the activity of which they advance through the soil or over the surface of the host, and lateral branches may originate from the inner cortex and also

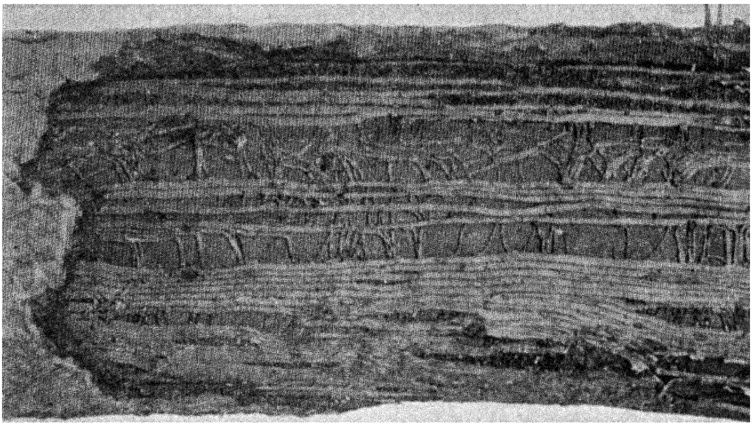

FIG. 239.—Rhizomorphs of *Armillaria mellea* between the bark and wood of larch stem. (*After Hiley.*)

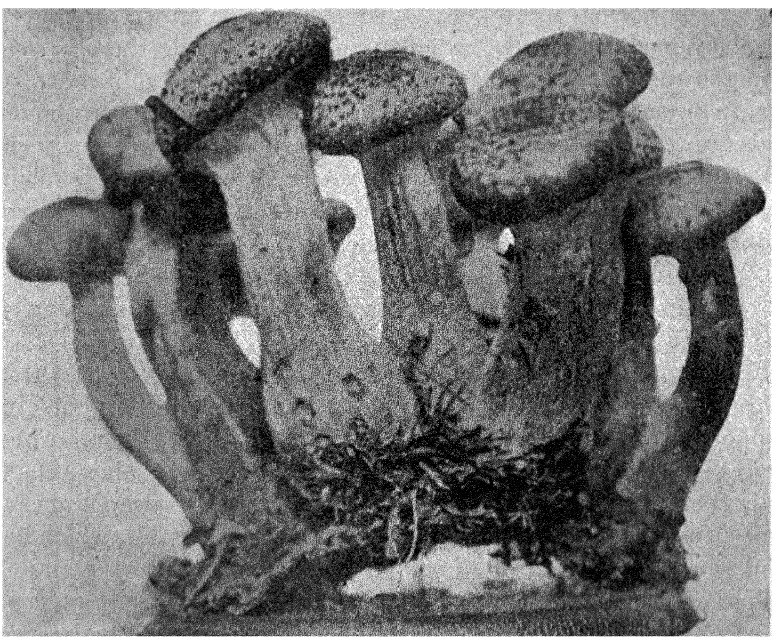

FIG. 240.—Group of nearly mature sporophores of *Armillaria mellea.* (*After Neger.*)

advance by an apical growth. At the apical region there is an external coating of loose hyphæ embedded in a gelatinous layer, but this disappears on the older parts.

The sporophores are produced in groups or clusters (rarely single) growing either from rhizomorphs or from cortical mycelial sheets, and generally appear around the trunk of the host close to the ground level, but in some cases may arise 6 feet or more up a trunk as in the Scotch or Austrian pines. These sporophores are found mainly during the few months of the year preceding the frosts of winter (September, October, November), but occasionally at other times (October to February in California); and are ephemeral structures lasting only until the spores have been disseminated. Each sporophore consists of a *stipe*, 3 to 10 inches long, honey-yellow or brown, bearing the expanded pileus which is honey-colored and sprinkled with dark-brown scales, while the lower surface is occupied by the whitish gills which are somewhat decurrent on the stipe. The sporophores are exceedingly variable in color, size

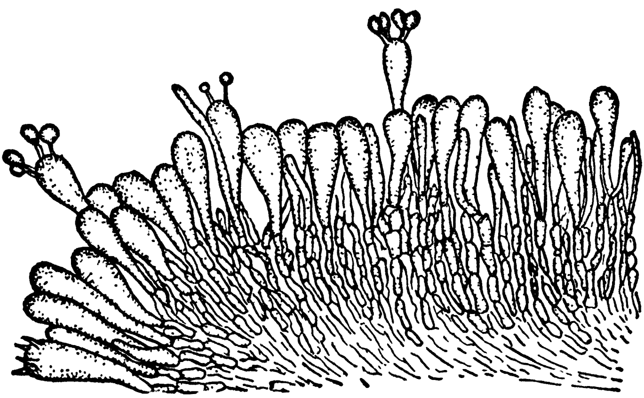

FIG. 241.—Small portion of basidial surface of *Armillaria mellea*. (*After Hartig.*)

and markings. The stipe is generally dark brown or black at the base, becoming lighter above, and marked by an *annulus* evident as a distinct membranous ring or reduced to a few delicate scales or even entirely absent. The stipe may be smooth, scaly or roughly grooved below the annulus, and may sometimes be more or less expanded at the base, rather than uniformly cylindrical. The pileus is at first distinctly convex, especially in the center, but with age may become flat, or even concave with an upturned margin, and is exceedingly variable in diameter (2 inches to 1 foot). In typical forms the scales are more numerous towards the center of the pileus and then thin out towards the periphery, become less evident with age and under certain conditions may be entirely absent. The lamellæ or gills, which are whitish at first, become flesh-colored or dingy with age. The hymenium consists of paraphyses and an abundance of *basidia*, each generally bearing four sterigmata (rarely two or three) with hyaline, elliptical or slightly reniform basidiospores, 6 by 9μ. These spores are forcibly detached and fall from between the gills, when they may accumulate as a white powder or be borne away by air currents,

the period of spore fall probably lasting several days. In a similar form Buller estimated that 40,000,000 spores fell from a single sporophore during each hour of the spore-fall period, which will serve to emphasize the fact that an enormous number of basidiospores are set free.

Sporophores are produced very abundantly from old decaying roots and stumps and from living hosts in the last stages of decline. Temperature and moisture conditions and the degree of disintegration of the substratum influence their development. In some cases the fungus may make only a vegetative development, producing mycelium and rhizomorphs without any fructifications. It is said that sporophores are rare in dense, dark, coniferous woods.

According to present opinions the spores and rhizomorphs play entirely distinct parts in the life of the root-rot fungus. It is the belief that direct infection of living structures by hyphæ formed by germination of spores in situ is relatively rare.

The spores are the common means by which the fungus attacks dead but uninfected stumps. A stump is employed as a base from which the fungus attacks living trees, so that spores are the medium by which it forms new *bases* from which the fungus proceeds on a career of penetration in limited tracts of forest (Hiley, 1919).

The rhizomorphs spread in the soil from their original bases (on dead roots or stumps) to adjacent living hosts, enter their roots or crowns through the open doorway of mechanical injuries or dead tissues and in some cases penetrate healthy, unbroken tissue. It has also been pointed out that xylophagous insects are important agents in the spread of the disease and the establishment of infections. New infections of living hosts take place then largely, if not entirely, through tissues in contact with the soil, and not through aerial structures.

Pathological Anatomy.—The mycelial fans or felts may be developed in the cambium, also in the outer phloëm or in the inner cortex, and in such cases several layers of the mycelium may be found, one inside another. The fungus does not advance far above the crown of the tree, the height varying with the host and the supply of moisture, from 2 to 5 feet or even more. From the cambial felts the hyphæ grow outward into the phloëm and inward into the wood, first following the medullary rays and spreading from these into the adjacent tracheides or vessels, and produce a white rot or disintegration of the wood. Early in the wood invasion black lines or layers appear.

These layers start from the cambium and gradually spread inwards, remaining very thin all the while, so that in transverse section of the trunk they appear as lines forming the sides of triangles with the cambium as the base. They also spread upwards in the wood, forming cone-shaped surfaces, and a section through a somewhat higher part of the trunk shows them as irregular circles (Hiley, 1919).

Black lines of this type are common in decaying wood, and White (1919) in his study of *Fomes applanatus* states "that cultural or other evidence shows that when they do occur more than one species of fungus is at work. They are produced at the point of contact of two invading fungi in the case of many pairs of wood-destroying fungi." Hiley attributed these black lines in mushroom root rot of the larch to the action of the single mycelium. According to this study, the hyphæ branch and become much segmented, the cells become bladder-like, their walls swell and become tinted with a pale-brown pigment, and closely pack the cavities of the tracheides. Some of these cells collapse and "their contents fill the interstices between the other bladders and stain the walls of the tracheides. Next the swollen hyphæ become bleached and empty, their walls again become thin and finally they disappear" (Hiley, 1919). The black line occupies a width of one or more tracheides and moves forward with the advance of the fungus. The writer cannot refrain from expressing the opinion that these inflated bladder-shaped bodies as figured by Hiley look like tyloses instead of like modified fungous cells, and the brown contents like "wound gum." Back of the black lines the tracheides "are delignified from the outside towards the middle lamella, leaving a layer of cellulose. Then the cellulose also is digested so that often whole walls disappear" (Hiley, 1919).

Host Relations.—Mushroom root rot is of importance as a disease of forest or shade trees, attacking both coniferous and broad-leaves species. It is stated that under European conditions it may attack any coniferous species that are making a poor growth. It has been noted especially on cedars, firs, hemlocks, larch, pine and redwood among the evergreens, and on alder, beech, birch, walnuts, almond, chestnut, locust, maple, mulberry, oak, sycamore and poplar among the broad-leaved trees. On the basis of studies in Oregon (Childs and Zeller, 1929) two different strains have been recognized: one on conifers, especially Douglas fir, which is not parasitic on apple trees; and the other originating from the native oak, *Quercus garyana*, which causes virulent infections on orchard trees but is not parasitic on conifers.

In America it is of most importance as a parasite of tree fruits: Apples, apricots, peaches, plums, prunes and cherries all suffer. The pear is claimed to be the most resistant of the tree fruits, the French pear being practically immune. Two other deciduous trees are reported as immune, the northern California black walnut (*Juglans californica hindsii*) and the fig (*Ficus carica*). Recent studies (Thomas, 1929) have shown no correlation between structural and morphological host differences and resistance. Resistance is believed to be a vital antagonistic reaction between host and parasite. "Of the three principal root stocks used for stone fruits, observations seem to indicate that myrobalan is the most resistant, surpassing either the peach or the almond root" (Hendrickson,

1925). Almonds, citrus fruits, olives and walnuts are seriously affected. Much injury has resulted to the grape in California and also in Europe. The bush fruits, especially in the Pacific Northwest, seem to be very susceptible, the greatest injury resulting to plantations of blackberries and raspberries. Horne (1915) has studied Armillaria in its relation to nursery stock, and believes that perfectly healthy nursery trees grown in Armillaria spots may become infected and thus carry the disease into the orchard, especially when "balled" trees, that is, trees with dirt, are transplanted. In the cut-over redwood lands of California the wild hazel is reported to be a favorite host of Armillaria (Essig, 1919). Many rhododendrons and azaleas in the city parks of Seattle were killed by shoestring root rot, probably *Armillaria mellea* (Schmitz, 1920) and more recently it has been reported as the cause of killing of flowering dogwood in the same city. It is also of interest to note that this fungus has been reported from Japan (Berkeley, 1922) as living in symbiosis with an orchid, *Gastrodia alata*.

The potato, carrot, parsnip, rhubarb, dahlias, cannas and strawberries may be mentioned as herbaceous hosts. Rhizomorphs may penetrate potato tubers and develop white, convoluted mycelial plates within the interior. One occurrence of this trouble in Washington was in the first crop on new land that had just been cleared of hazel brush.

Control.—The problem of prevention of root-rot injury is somewhat different for forest trees than for orchard crops. In Europe the recommendation is made that infested wood lots be cleared and devoted to the growth of some farm crop for a time, and that coniferous plantations should not follow immediately on land from which broad-leaved trees have been cleared. In America the disease in fruit trees and other cultivated hosts seems to be due largely to planting in cut-over land in which the fungus was already established either as a saprophyte or a parasite of the native trees or shrubs. Experience has shown that Armillaria will disappear from cleared land after a period devoted to some non-susceptible farm crop, hence in selecting sites for orchards newly cleared lands may be viewed with suspicion, and planted to a non-susceptible crop if Armillaria is known to be present.

The disease frequently appears in established fruit plantations, and then the problem is one of either saving the affected trees or of preventing the spread to adjacent healthy trees, or making replanting safe. The practices to which consideration should be given are as follows: (1) The removal and destruction of diseased trees or plants, including as much of the root system as possible as soon as the disease is discovered. This practice should be followed at least when the host is seen to be very seriously affected. (2) The removal and destruction of sporophores when in the button stage, so as to prevent the maturing and dissemination of basidiospores. Diseased or dead trees or stumps should be watched

for the appearance of the characteristic groups of fructifications. (3) Since the fungus is known to spread in the ground by means of its rhizomorphs, the construction of barriers has been recommended to confine them and keep them from reaching other tree roots. The common barrier suggested is a trench 1 foot wide and 2 feet deep, with the dirt thrown towards the center, surrounding a single tree or groups of diseased trees, and so located as to be beyond the spread of the roots. Horne has also suggested the use of a concrete wall as a barrier. If an adjacent stump appears to be the base from which the fungus originated, it should also be enclosed by the barrier. (4) The possible treatment of diseased trees when the disease is discovered in its earlier stages, with the idea of either saving the tree or prolonging its life. The treatment of apple trees in Oregon has given some promise of success (Barss, 1913) and consists of removing the soil from around the crown for a foot or more so as to expose the large roots, to be followed by the cutting out of diseased bark or roots. The cut surfaces are then disinfected with creosote, or Bordeaux paint, and protected with a waterproof wound dressing. If the trunk lesions are of great extent, bridge grafting may be needed to assist in the recovery. The exposed crown and roots are to be left exposed to the air and sunlight during the summer, but the dirt should be thrown back before freezing weather. This surgical treatment, followed by the prolonged aeration of the crown, has been tried for orange trees in Australia, but gave poor results unless the trees were only slightly infected when the operation was performed. (5) Delay in resetting of root-rot spots or places from which individual diseased trees have been removed or replanting with a highly resistant variety. Three years is supposed to be the minimum period of rest unless the soil can be treated with chemicals to kill the rhizomorphs. At present specific and reliable information concerning chemical treatment is not available.

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IMPORTANT TROUBLES DUE TO PALISADE FUNGI

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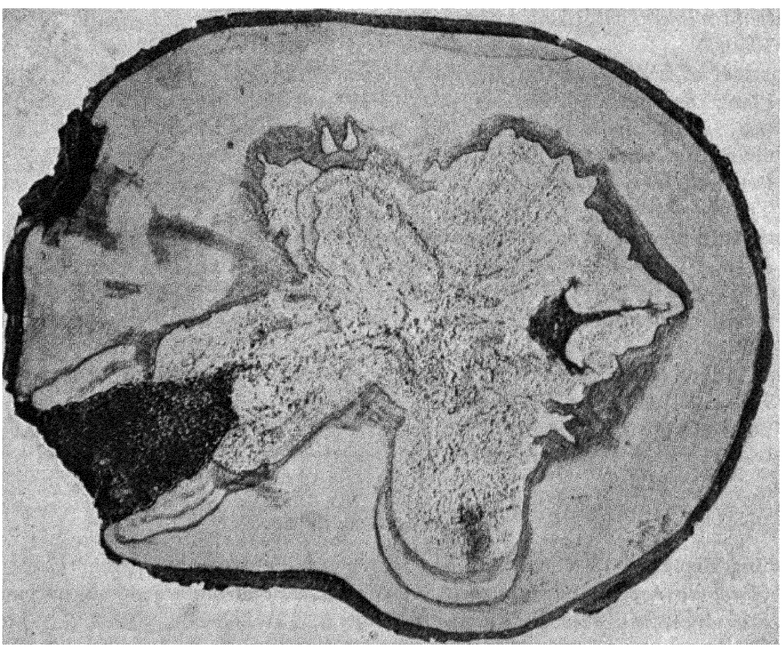

Fig. 242.—Cross-section of the trunk of a living silver maple rotted by *Fomes igniarius*. (*After von Schrenk and Spaulding, B. P. I. Bul.* **149**.)

af Slaegten Merulius (Hall) Fr. med en saerlig Omtale af Gruppen Coniophori Fr. *Dansk. Bot. Arch* **5**: 1–47. 1928. Hubert, E. E.: *Loc. cit.*, pp. 473–484. 1931.

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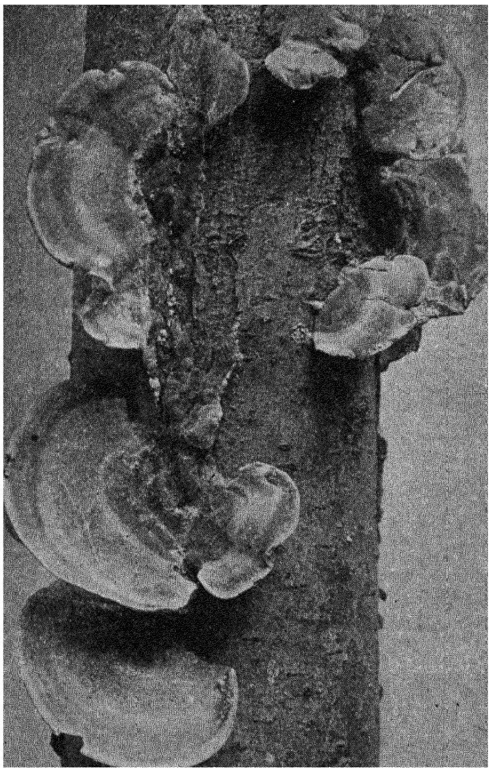

FIG. 243.—Portion of trunk of mountain ash with sporophores of *Polystictus hirsutus*.

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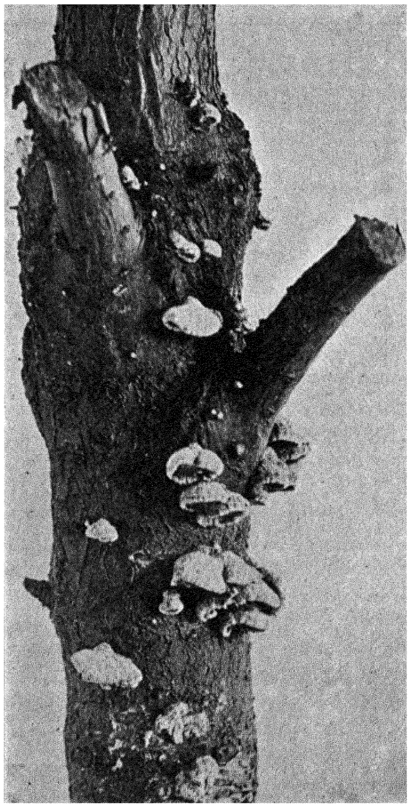

Fig. 244.—Portion of trunk of a young cherry tree invaded by *Schizophyllum alneum.* The fungus entered through the long stubs left in pruning.

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CHAPTER XXVII

PARASITIC SEED PLANTS AND THE TROUBLES THEY CAUSE

PARASITIC SEED PLANTS

Many flowering or seed-bearing plants are parasites or half parasites. The latter have a chlorophyll apparatus, while the former have none. These parasitic seed plants cause more or less disturbance in the life of the hosts which they parasitize and, when crop plants are concerned, may cause serious injury. Before considering the groups of parasitic seed plants the types of nutrition of seed plants in general will be briefly outlined, the robber class constituting one of the five types.

Types of Nutrition of Seed Plants.—From the standpoint of their nutrition, seed plants show a considerable diversity. They may be grouped as follows:

1. The *factory owners*, the majority of green plants, which by means of their chlorophyll apparatus are the producers of the world's food supply (holophytes).
2. The *profit sharers*, those which have a mutualistic relation with some other organisms, either bacteria or fungi (symbionts).
3. The *trappers*, plants with ingenious devices for capturing insects which they digest (insectivorous plants).
4. The *scavengers*, chlorophyll-less plants, which have become partially or completely saprophytic in their mode of life (saprophytes).
5. The *robbers*, those which steal a part or all of their food from other plants, their hosts, and are therefore either semiparasitic or completely parasitic (parasites).

Groups of Parasitic Seed Plants.—Of the total number of seed plants, only a few have degenerated to the robber class, and of these only a few are of importance because of their injurious relations to plants of economic importance. They exhibit, however, many interesting parasitic relationships. These parasites are to be found in a small number of families or subfamilies, some of which show little morphological relationship, but frequently marked physiological similarities. The following groups may be recognized:

1. Herbaceous plants bearing green foliage leaves, and rooted in the soil, but provided with haustorial disks, which are attached to the roots or crowns of their hosts. This type of parasitism is to be found in 100 or so species of Santalaceæ, represented by the true sandalwood (*Santalum*

album) of India, bastard toadflax (*Thesium alpinum*) of Europe and *Commandra spp.* attacking various woody and herbaceous hosts; also in many Rhinanthaceæ, some of the better known species being the eyebright (*Euphrasia spp.*), the yellow rattle (*Rhinanthus spp.*), cow wheat (*Melampyrum*) and lousewort (*Pedicularis spp.*). Most of these parasites cause little if any appreciable injury to the host plants with which they are associated.

2. Underground plants, with perennial stems, bearing leaves devoid of chlorophyll and with haustorial disks attached to the roots of trees and shrubs in the same manner as in the first group. Each year aerial flower-bearing shoots are formed which die down with the ripening of the seed. The toothwort (*Lathræa squamaria*), the best known representative, in addition to obtaining part of its food supply from its host, has unique leaf traps by which it captures minute earth-inhabiting animal organisms and utilizes them as food. These species also belong to the Rhinanthaceæ.

3. Foliage-bearing, chlorophyll-containing, bushy, perennial plants growing upon the aerial portions of various trees. The seeds germinate on the branches of the host and soon establish a nutritive relation by the penetration of haustoria. Some Santalaceæ of the East Indian Archipelago belong to this group, but the best-known forms belong to the Loranthaceæ. The most important representatives are the giant mistletoes (*Loranthus spp.*), mainly tropical parasites, but including *L. europæus* on oaks and chestnuts in the east and south of Europe; the European mistletoe (*Viscum album*); the American mistletoes (*Phoradendron spp.*); and the dwarf or scaly mistletoes (*Razoumofskya spp.*). The last are especially injurious to coniferous trees in the northwestern United States (Fig. 252). There are distinct species which parasitize the western larch, lodgepole pine, western yellow pine and Douglas fir.

They may kill young trees or older trees may be retarded in growth of both height and diameter. Severe infestations may also cause the death of upper branches, producing staghead or spike tóp, while large burls, or witches' brooms, may be formed which interfere with the life processes of the tree (Weir, 1916).

4. Twining plants with filiform stems devoid of green leaves and without chlorophyll, but with a few rudimentary scale leaves still persisting. The plant retains no connection with the soil but twines around its host from which it absorbs its crude and elaborated food by means of root-like sucking organs or haustoria. The group is represented by two entirely unrelated genera: Cassytha, with mostly tropical species (family Lauraceæ) of the eastern and western hemisphere, with a single species (*C. filiformis*) in the rosemary scrub of Florida; and Cuscuta (family Cuscutaceæ), with numerous species in various countries. These dodders or love vines are of outstanding importance because of their disastrous

effects upon various cultivated plants, especially clovers and alfalfa (see special treatment).

5. Plants destitute of chlorophyll, and with seeds that germinate in the soil where a filiform plant body is formed which grows into the ground and soon penetrates crown or root of its host. Its tissues are merged with those of its host to form a more or less tuberous enlargement, from which aerial flowering stems arise at some later time. These flowering shoots may be almost naked, clothed with a few scattered rudimentary leaves, or covered with conspicuous, closely imbricated scale-like leaves. The most important representatives of this group belong to the broom rapes (Orobanchaceæ) and the Balanophoraceæ.

The broom rapes include 130 or more species, mostly from north temperate regions with a few from the tropics and the southern hemisphere. Several species of broom rapes are of economic importance. The best known are the branched broom rape (*Phelipœ ramosa*), parasitic on the roots of tobacco, hemp, etc., and *Orobanche minor* on clovers and some other hosts. The broom rapes are more serious in Europe than in America. In its maximum development, the clover broom rape (Kleeteufel) may be so abundant as entirely to destroy the second cutting. Some of the species are small, but several natives of North Africa reach a height of 50 centimeters and produce stems as thick as one's arm.

The Balanophoraceæ are found mainly in an equatorial belt encircling the world, and inhabit primeval forests, where they are parasitic on the roots of woody plants which run below a covering of vegetable mold.

6. The representatives of this group show the most extreme merging of the plant body of the parasite with the tissues of the host. The young plant penetrates the cortex of the host and finally forms a more or less definite hollow cylinder, which is intercalated between the wood and the cortex. The stem or root attacked shows little or no enlargement at the place where the parasite is located, and the infection is first evident when the flowers burst through the cortex. These forms belong to the single family Rafflesiaceæ, which has representatives in the tropical and subtropical regions of Asia, and adjacent islands, tropical America and subtropical South America, and a single species, *Cytinus hypocistus*, in Mediterranean Europe. The genus Rafflesia is noteworthy as including a species *R. arnoldii*, which produces flowers 1 meter in diameter, claimed by some authorities to be the largest flowers in the world. This wonderful flower is a native of Sumatra and is sessile upon roots of vines. The open flower displays five immense fleshy lobes around a central bowl-like portion within which the stamens and styles are inserted. The central bowl and its surrounding fleshy ring or corona are blood red, while the lobes are nearly the color of the human skin, and the flower emits a putrescent, cadaverous smell. Another species of nearly equal

size, *R. schadenbergii*, is found in Mindanao, of the Philippine group. A single flower may weigh about 22 pounds.

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DODDER OR LOVE VINE

Cuscuta spp.

The dodders or love vines are non-chlorophyll-bearing, leafless, twining, parasitic seed plants which attach their yellow, orange or pink, thread-like stems to the stems or other parts of various cultivated or wild plants, sometimes on single isolated hosts, but frequently as conspicuous tangles of intertwining stems. Various other common names more or less descriptive of these parasites are in use in various localities, some of the more important ones being "strangle weed," "gold thread," "hair weed," "pull-down," "hail weed," "devil's hair," "devil's ringlet," "devil's guts" and "hell-bind." Dodders are of much importance as pests of clovers, alfalfa and flax, but are of minor concern as parasites of various other cultivated plants.

American Dodders.—These parasites all belong to the genus Cuscuta of the Cuscutaceæ or dodder family, which was formerly considered a tribe or subfamily of the Convolvulaceæ or morning glory family. We still consider the dodders as very close relatives of the morning glories, although they have undergone marked physiological and morphological changes as a result of their parasitic mode of life. The first American monograph of the genus was published by Engelmann in 1842, and this was elaborated in a later publication (1859). In the recent "Revision of the North American and West Indian Species of Cuscuta," by Yuncker (1921), 54 species and 42 varieties are described. The most important of the species from their relation to our cultivated crops are as follows:

1. *Clover Dodder* (*Cuscuta epithymum* Murr.).—This species attacks various species of clover, alfalfa and some other legumes. It is an introduced species, occurring throughout North America, but does little damage after the first season, as it rarely produces seed in the United States.

2. *Small-seeded Alfalfa Dodder* (*C. planiflora* Ten.).—This is another emigrant from Europe, which is found on alfalfa and some other legumes, mainly in the western states from Colorado, Wyoming and Washington south to New Mexico.

3. *Large-seeded Alfalfa Dodder* (*C. indecora* Choisy).—This native species shows a preference for leguminous crops, especially alfalfa, and is common from Colorado westward. It is less common in the South and has been sparingly introduced into eastern states. Yuncker recognizes five varieties (1921).

4. *Chilean Dodder* (*C. racemosa chileana* Engelm.).—This South American species has been sparingly introduced and attacks both clover and alfalfa. It has been reported from widely separated stations from Maryland to California. It is common in red-clover and alfalfa seed imported from South America.

5. *Flax Dodder* (*C. epilinum* Weihe).—This European species attacks flax and sometimes other hosts but never clovers or alfalfa. It is limited largely to the flax-producing sections of this and foreign countries. None of the other species of economic importance attack flax.

6. *Field Dodder* (*C. pentagona* Engelm.—*C. arvensis* Bey.).—This native species is widely distributed throughout North America, but is most common and serious east of the Mississippi River. It infests many species of herbaceous plants, but shows little preference for any special hosts, cultivated or wild. It is not uncommon on clovers and alfalfa, and has caused serious injury to the sugar beet. Yuncker recognizes four varieties (1921).

7. *Common Dodder* (*C. gronovii* Willd.).—This is another native species with little preference as to the plants which it attacks. It is

reported as infesting garden ornamentals and even hedge plants or willows.

The various species of dodder are so very similar in appearance that no attempt will be made to offer diagnostic characters even for the economic species, but the student is referred to taxonomic manuals or to Yuncker's monograph for detailed descriptions. It may be noted that

FIG. 245.—Dodder on alfalfa.

alfalfa and clover are the principal crop plants which suffer from dodder attacks, certain species showing a decided preference for these hosts. Grass or cereal hosts are never seriously affected, but may be parasitized by the less specialized species when growing in mixed cultures of preferred hosts.

The Parasite.—The various species of dodder are so similar that a single description of their general characters will suffice. When dodder

first becomes evident in a field it will be noted as a tangle of branched, thread-like, leafless stems, devoid of chlorophyll or green pigment, twining around the stems or other parts of its host, or forming an interlacing mat. The common color is yellowish or orange, but the stems of certain species

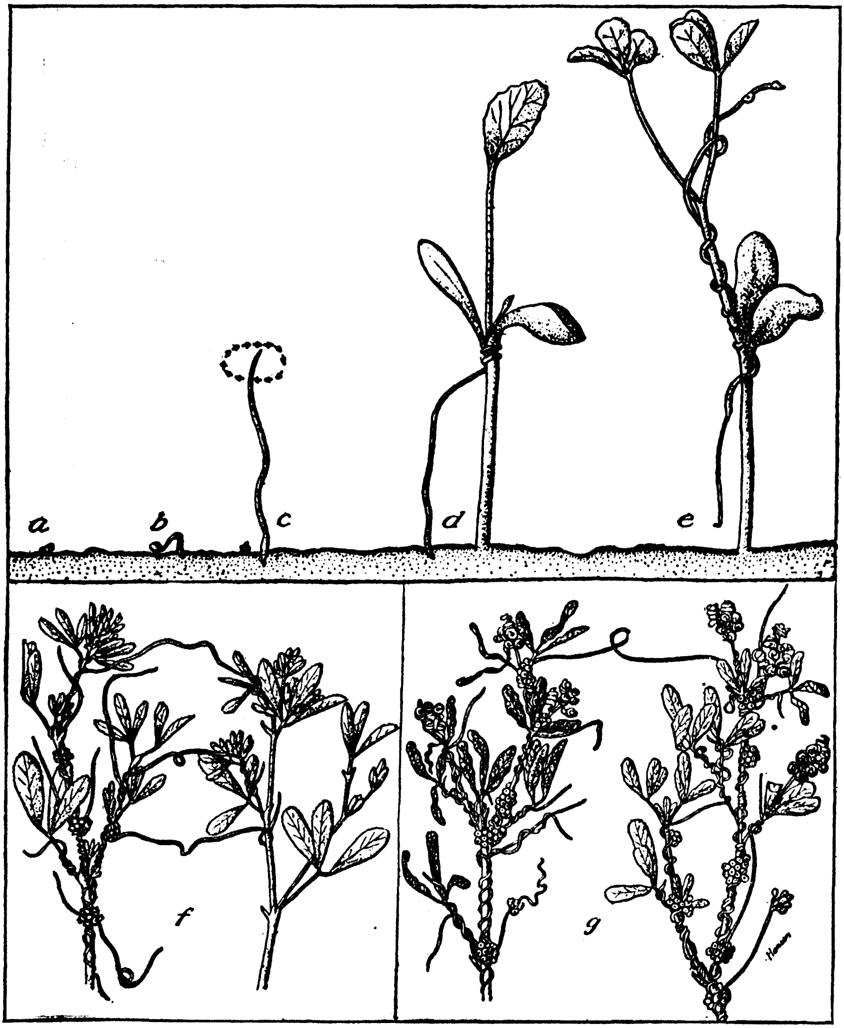

FIG. 246.—Diagrammatic representation of the complete life cycle of dodder from the seed (*a*) to the maturing of a new crop of seed. (*After Hansen, U. S. Dept. Agr., Farmers' Bul.* **1161.**)

are frequently tinged with red or purple, or may in other cases be almost white. While true leaves are absent, they are represented by minute functionless scales, which are evident on close examination. Whenever the twining stems come into contact with the host, minute root-like organs, the *haustoria*, penetrate into the host cortex and serve for the

absorption of both crude materials and elaborated food. The established dodder plant has no connection with the soil but derives its entire supply of nourishment directly from its host.

The tiny, white, pink or yellowish flowers occur in clusters, appearing from early June until the end of the growing season. The flowers are mostly gamosepalous, and pentamerous; the stamens inserted in the throat of the gamopetalous corolla, alternating with the lobes; a fringed or fimbriate structure usually present below each stamen; ovary two-loculate, each cell with two ovules; the two styles distinct or united and with capitate or linear stigmas; fruit a capsule, producing tiny gray or reddish-brown, slightly roughened seeds. The seeds ripen from July

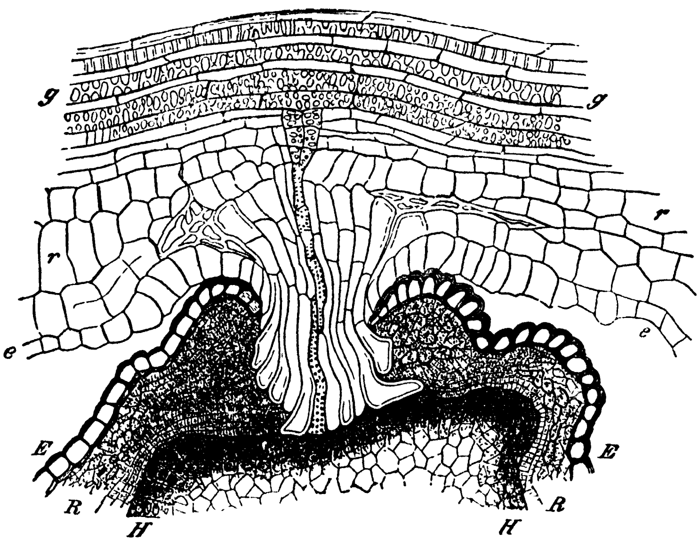

FIG. 247.—Haustorium of *Cuscuta epilinum*. *E*, epidermis; *R*, cortex; *H*, wood of flax stem; *e*, *r* and *g* are tissues of Cuscuta. Semidiagrammatic. (*After Sachs.*)

until frost and under favorable conditions are produced in great abundance. A single plant has been reported to produce as many as 3000 seeds.

Under favorable conditions of moisture and temperature the seeds of dodder germinate like any other seed and produce young seedling dodder plants. It has been noted that the seeds of alfalfa or clover dodders generally germinate in the spring a few weeks later than the seeds of their hosts. A young seedling is a slender, yellowish, unbranched thread, generally larger at the lower end, which may remain for a time either in contact with the soil or slightly embedded in it. The distal end is raised into a more or less vertical position and the growing tip describes a circle in its search for a support. When a support is reached, whether it is a congenial host or not, the young stem begins to twine around it, the same as a morning glory around its support. If the supporting structure is a

susceptible host, the young stem soon forms haustoria, which penetrate the host tissue, and infection is accomplished. From now on the young seedling has an abundance of available food and the young stem below the first stem coil and haustoria shrivels and dries up, and all contact with the soil ceases. If a young seedling does not establish nutritive relations with a host it perishes as soon as it has used up the food supply that was stored up in the seed. Seedlings of alfalfa dodder have been found to retain their vitality for 6 to 9 weeks without establishing a host connection. The established parasite continues to climb upward, and "waxes fat" at the expense of its host, while the stem which was simple at first develops branches and the twisting or circumnutating tips reach out and twine around adjacent host parts. In this way an increasing zone of infection results.

The origin and development of the haustoria may be briefly noted. They appear on the concave side of the stem coils as a result of the contact stimulus.

A sucker-like organ first arises from the epidermis of the mother stem and adheres firmly to the host plant; its formation is then followed by the ingrowth of the true haustorium, which has originated endogenously, mainly from the cortical region just outside the pericycle, and seems very properly to represent an adventitious root. The cells of the sucker, or "prehaustorium," dissolve their way into the host plant, partly by pressure, partly by the excretion of a ferment, and into the space thus made the haustorium grows, enlarging the opening and becoming surrounded by a mass of compacted dead cells (Thoday, 1911).

The superficial cells of the developing haustorium become greatly elongated to produce a bundle of hypha-like cells, which advance inward. Omitting some of the details of development that are unessential for understanding the relation of the parasite and host, it may be noted that some of the central cells push on through the vascular cylinder into the pith, while the elements immediately around the central core become applied to the xylem elements of the host. The inner peripheral cells of the haustorial brush penetrate to the functional and developing sieve tubes of the phloëm and spreading outward come into intimate contact with sieve tubes of the host. The haustorial cells coming into contact with the host xylem elements organize strands of tracheids which make a continuous connection with the xylem elements in the main stem of the parasite, while those in contact with the sieve elements of the host organize strands of sieve tubes which form a continuous cylinder connecting with the phloëm elements of the main stem. An effective parasitic relation is thus established with both phloëm and xylem of host and parasite in physiological continuity, thus affording a pathway along which both crude and elaborated food may be transported from the host stem into the stem of the parasite.

Effect of Dodder.—In a crop like clover or alfalfa the dodder usually appears in circular spots, the size depending upon the age of the infestations, those of a single season's growth varying from 3 to 6 feet in diameter.

Usually, they increase in size from year to year, ultimately reaching a diameter of 30 feet or more. In some spots the dodder dies out and may be said to have become extinct. By the coalescence of two or more spots, large bare areas of irregular shape are formed. On the interior of the spots there remain a few scattered alfalfa plants which, somehow, escaped destruction by the dodder, but the ground is occupied chiefly by weeds (Stewart, 1908).

In old-established infections, the dodder is not much in evidence in the early spring, but later it develops the characteristic tangled masses of the yellow dodder stems on the host plants around the margin of the spots. The amount of damage in alfalfa or clover fields will vary with the species of dodder, the host conditions and the amount of seed of the parasite that was introduced into the field. Cases are on record in which entire fields have been ruined during the first year, but generally the injury is less severe. The maximum injury in most cases results during the second or third years after seeding. Dodder-infested fields may have the hay crop lowered in quantity and quality, but dodder is of most concern in seed-producing regions. Although dodder is a noxious parasite,

. . . it is not to be feared in the United States in the same degree that it is dreaded in Europe. In parts of Europe, especially Germany, the production of clover seed has ceased because of the ravages of dodder. The conditions in this country do not seem to be as favorable as in Europe for the development of dodder; hence the discovery of dodder on the farm should not be the occasion for serious alarm, but rather for the employment of a well-conceived and systematic plan for its extermination (Hansen, 1912).

Etiological Relation.—Dodder overwinters by means of seed which falls to the ground and remains dormant until spring, or in the case of certain species on perennials, by means of portions of its stems which are resistant to winter temperatures. While many botanical writings have classed the dodders as annuals, it has been shown by Stewart and others (1909) that certain species—*C. epithymum* on alfalfa, for example—are perennial and that the continuation of infestations is more often due to the resumption of growth by overwintered stems than to the germination of new seed. In fact, in the United States this species produces seed only rarely.

Attention may be directed to the habit of growth which makes the extermination of dodder in a crop like alfalfa difficult. If dodder were rooted in the soil like an ordinary weed the problem would be much simplified, but it is really rooted by means of its haustoria at numerous points, where its stems coil around the host parts. The various portions of the parasite are therefore independent, and even minute pieces of its

stem can continue growth when connection with the parent plant is severed, provided there is a haustorial contact with the host. Even close cutting of alfalfa leaves sufficient crown infections to continue the growth of the parasite on later crops. It has also been shown that separated fragments of dodder stems a few inches long may, under moist conditions, establish new haustorial connections if left in contact with susceptible hosts.

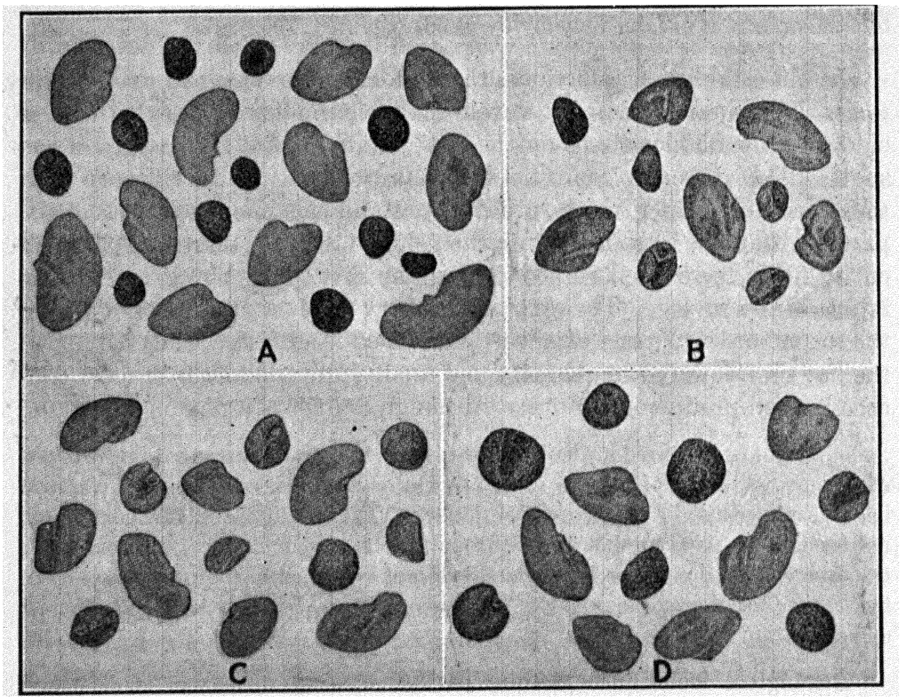

FIG. 248.—Seed of dodder compared with alfalfa seed. *A*, clover dodder (*Cuscuta epithymum*); *B*, small-seeded alfalfa dodder (*C. planiflora*); *C*, field dodder (*C. arvensis*); *D*, large-seeded alfalfa dodder (*C. indecora*). (*After Hillman, U. S. Dept. Agr., Farmers' Bul.* **306**.)

Dodder may be introduced into a field, or when once introduced it may be disseminated by any one of the following methods: (1) as impurity in the seed; (2) by irrigation water; (3) by hay from infested fields; (4) by manure containing the seeds; or (5) by farm operations and the movement of live stock from one field to another. The presence of dodder in commercial seed of clovers or alfalfa is due to the presence of dodder in seed-producing sections, and not ordinarily to the adulteration of the seed, although screenings from low-grade seed may sometimes be mixed with other seed by unscrupulous dealers. The size of dodder seed is such that it is not removed in the threshing operation, and so is left in the commercial seed unless some special method of separation has been prac-

ticed. It has been noted that half-ripe dodder seed will germinate as readily as fully matured seed, and that dodder which has not yet formed seed at time of cutting may still obtain sufficient food to ripen its seeds. While dodder-containing seed is the principal source of the first introduction of the parasite into a field, its further dissemination may be brought about by some of the other agencies.

Control and Eradication.—The dodder pest can be handled by following practices to prevent its introduction and, if present, to guard against further dissemination; or established infections may be eradicated. The important preventive measures are as follows: (1) the avoidance of dodder-infested seed; (2) the avoidance of dodder-infested hay; (3) the exclusion of grazing animals from dodder-infested fields, or at least preventing their movement from infested fields to clean fields; (4) the eradication of dodder from localities may contaminate irrigation water and thus carry the seed from place to place; and (5) the avoidance of dodder-containing manure unless this has been composted for 6 or more weeks.

It is of first importance to avoid the purchase of dodder-infested seed. Many states require commercial seed of clovers or alfalfa to be labeled as to purity or dodder content, while the Federal law excludes the importation from foreign countries of commercial seed containing one or more dodder seeds per 5 grams of seed. These measures have improved the quality of seed upon the market, but still great care should be exercised in selecting seed to be sure that it is free from dodder.

Dodder seeds range in size in the different species from those slightly smaller than the seed of white clover to those as large as or larger than alfalfa seed.

In general, these seeds are usually *dull coated, with roughened,* or *minutely pitted surfaces,* and each seed has three flat surfaces, while the seeds of clovers are usually smooth, rounded and possess a certain luster. Furthermore, the scar on dodder seeds is very inconspicuous, whereas the scar on leguminous seeds is clearly evident. Dodder seeds range in color from dark brown to green or yellow (Hansen, 1921).

The grower who has not acquired the ability to recognize dodder seed should have his seed tested by either a Federal or a State seed laboratory.

The removal of dodder seed from large lots of seed can be done most economically and effectively by seed companies having special cleaning machinery. A high per cent of the dodder seeds can be removed from clover or alfalfa by power-driven or hand graders, with the use of the proper screens. Recently excellent results have been obtained with the Dosser machine, in which the velvet linings remove many small dodder seed that cannot be screened out. Still more recently, an electromagnetic process of separation has been devised. The seed is mixed with a powder which is sensitive to a magnet and more adheres to the rough dodder seeds than to the smooth legume seeds. When passed

under a magnet the heavily coated seeds are withdrawn, and these include not only the dodder, but some weed seeds and fragments of legume seeds. After the separation the legume seed is passed through a polisher to remove any adhering powder (Foy, 1924).

Dodder seed may be separated from clover or alfalfa on the farm by hand sieves. Stewart (1909) recommends a foot-square frame 4 inches deep, with a screen made of 20 by 20 mesh, using either No. 34 Washburn and Moen gage, steel or iron wire, or No. 33 brass or copper wire on the Old English gage. One-fourth to one-half pound of seed is to be shaken vigorously in the screen for ½ minute. Robbins and Egginton (1918) recommend a 20 by 22 mesh brass screen, using 32 or 34 gage. Hansen (1921) says: "Best results have been obtained by the use of a sieve made of wire having 20 meshes to the inch and of No. 30 to 34 thickness." The large-seeded alfalfa dodder cannot be removed by these screens, hence seed infested with this species should be discarded.

The success of eradication measures and the plan to be followed will depend upon the extent of the infection, that is, whether only small isolated areas are present or whether general infestation prevails, and also upon the seed formation by the parasite. In either case eradication measures should be initiated, if possible, before seed is formed. The methods of treatment are as follows: (1) Small scattered patches before seeding. Mow and allow the plants to dry, or sprinkle with crude oil or kerosene and burn, or feed the cut plants for hay. (2) Small scattered patches, after seeding. Burning by one of the following methods is recommended: (*a*) Mow close, beginning at the outer margin of the spot and work towards the center. Allow the plants to dry, then burn. (*b*) Sprinkle with kerosene or crude oil and after a few days ignite. (*c*) Cover infested area with straw and burn. (*d*) Burn the infested plants with a blow torch. (3) General infestations before seeding. In this case the crop may be mowed early, at least before any of the flowers of the dodder open. Close grazing by sheep or hogs or plowing the crop under for green manure may be a substitute treatment. (4) General infestations after seeding. In cases of severe infestation with dodder, cut close to the ground, and allow the plants to dry, or sprinkle with crude oil or kerosene and burn in the field. Fallow for the remainder of the season or plant a winter crop, and then follow with a 5-year rotation, beginning with a non-leguminous, tilled crop. At the end of 5 years. if non-susceptible crops have been used, it will be safe to seed to clover or alfalfa again. To avoid loss of the hay crop, it may be cut and fed to live stock without removing from the field, and during following years the crop should be cut before the dodder blossoms, or close grazing practiced until the dodder is under control.

If general field or garden crops are attacked by dodder and the infestation is severe, the practical solution is to grow either resistant or immune

varieties until the dodder disappears. It should be noted that new lands on which dodder has been prevalent may have their first crop infested with this parasite. This is not a common occurrence, but it should be recognized as a possibility.

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THE AMERICAN MISTLETOES

Phoradendron spp.

These leafy, green parasites of forest and shade trees are a familiar sight from New Jersey, southern Illinois and Oregon southward, while beyond their natural range, they are frequently found upon the market during the Christmas season, and in many homes constitute a part of the holiday decorations. In some sections of the country certain species are very prominent because of their serious injury to forest and shade trees.

History and Geographic Distribution.—The common mistletoe of Europe is *Viscum album*, and it was at first supposed that the common mistletoe of the Atlantic Coast (*P. flavescens*) was identical with the European species. Although the American leafy mistletoes were at first generally referred to Viscum, they were early recognized as showing marked differences, but were not separated until the pioneer botanist, Nuttall, in 1847 placed them in a new genus to which he gave the name of Phoradendron, meaning tree thief. The genus is strictly American, the various species ranging from Oregon, southern Colorado, the mouth of the Ohio River and southern New Jersey southward into Mexico, Central America, the West Indies and in South America to the mouth of the La Plata, while two species are found in the Galapagos Islands of the Pacific and one in the Pacific island of Guadalupe. A late monographic revision of the genus by Trelease (1916) recognizes 277 differentiable forms, of which 240 are regarded as species. The genus is divided into two groups distributed as follows:

	United States	Mexico	Central America	West Indies	South America	Total
Boreales	28	48	2	0	0	78
Æquatoriales	0	29	20	38	134	221

Of the species occurring in the United States a relatively small number have a wide distribution. *P. flavescens* Nutt., which has been called the American mistletoe, "occurs from southern New Jersey to the lower Wabash, Oklahoma and eastern Texas, reaching southeast to the gulf and ocean" (Trelease, 1916) and parasitizes a great variety of Angiosperms. *P. villosum* Nutt., the Pacific Coast or California mistletoe, is found from Oregon south through California and lower California, usually on oaks but also on numerous other genera of Angiosperms. *P. californicum* Nutt., the legume, red-berried mistletoe, is found in California, Utah, Arizona and lower California, chiefly on Leguminosæ, but never on Coniferæ. In the southern area between the ranges of these species *P. engelmanni* Trel. is the important species of central and western Texas, while *P. macrophyllum* Cock. is common in Arizona and to less extent

in adjacent Mexico. While the genus is prominent as furnishing parasites of Angiosperms, several species parasitize coniferous hosts, notably *P. juniperinum* Engelm. on Juniperus in southern Colorado, Arizona, New Mexico, western Texas and adjacent Mexico. The mistletoe has attracted the most attention as a pest in Texas and other portions of the Southwest (York, 1909). It has been noted that in the transition zone from the humid climate of the Gulf states to the arid climate of the Southwest, in which conditions are unfavorable for the best tree growth, mistletoe flourishes, and is more varied in form and relatively more abundant than in the more humid areas. In explanation of this behavior it has been suggested that "mistletoe, like a good many other plants of arid situations, requires much sunlight for its best growth, and especially for the development of flowers, and thereby of numerous and vigorous seeds, and is at a disadvantage in competing with the heavy shade-casting foliage of forests in humid climates. The necessity for light might explain why in bottom-land forests of the East mistletoe is confined to the highest branches of the tallest trees, and how, with increasing intensity of sunlight and the more meager foliage and open stand of trees incident to the drier climate of the Southwest, mistletoe is enabled to spread over the entire tree" (Bray, 1910).

The Parasite.—Most of the species of Phoradendron show a marked similarity in growth and general habit, appearing as bunched tufts of leafy, perennial, suffruticose shoots on the branches of their hosts. Because of their evergreen character, they present a striking picture, when their hosts are devoid of foliage. The European Viscum and the American Phoradendron are so similar in general appearance that a winter picture of either might easily be mistaken for that of the other.

A marked exception is found in some of the desert mistletoes, like *P. californicum* and *P. libocedri*, which when seen from a distance sometimes suggest the cactus genus *Rhipsalis* in their long pendant tufts: and the Mexican *P. calyculatum* and a few other species form wide-spreading, fountain-like masses of still greater size (Trelease, 1916).

The shoots are well supplied with opposite, expanded leaves, which are petioled or petiolately contracted, but in a few species they are reduced to scales. There is a well-developed chlorophyll apparatus in both leaves and stems, by which the parasite is able to manufacture its own carbohydrate food, but the plants, especially during the winter, have a slightly yellowish-green cast, in some species even a golden coloring (*P. macrophyllum*) or an olive or brownish shade (*P. juniperinum*, etc.). The stem of the mistletoe expands in the cortex of the host into an irregular branched structure, the *haustorium*, from the under side of which peg-like outgrowths, the sinkers, are formed which penetrate to the cambium and later, by the formation of the annual rings of xylem, come to be embedded in the wood.

The haustorium and sinkers fix the parasite in position and serve as an absorbing organ. The sinkers contain no phloëm elements, and only come into direct contact with xylem cells of the host, hence the parasite cannot rob its host of elaborated foods, which must travel along the phloëm. The mistletoe is then what may be called a *water parasite*,

that is, it obtains its water and mineral salts from its host, but elaborates its own food in the same way as an ordinary green plant. The hausto-

FIG. 249.—A typical growth of *Phoradendron engelmanni* about 2 feet in height. (*After York.*)

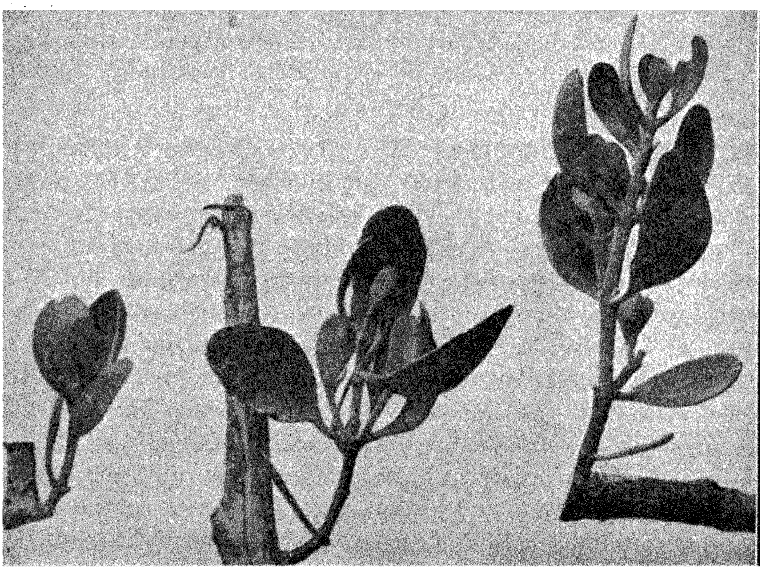

FIG. 250.—Mistletoe plants 5, 6 and 7 years of age. (*After York.*)

rium gives rise to buds on its upper surface, which may produce new shoots at some distance from the primary shoot. If the aerial shoots are

broken off, the cortical haustorium will immediately give rise to new shoots from dormant buds which are started into activity.

The northern mistletoes are diœcious, with axillary spikes of 3- or sometimes 2-, 4- or 5-merous, inconspicuous, apetalous flowers. The sepals are distinct, deltoid, valvate and persistent on the fruit; the stamens on the base of the sepals, with sessile, two-celled anthers opening by subapical slits. The ovary is inferior, one-celled, one-ovuled and develops into a more or less globose berry with a single albuminous seed and a very viscid mesocarp. The mature berries of the common species are whitish translucent, or sometimes shaded with greenish yellow, while the legume mistletoe (*P. californicum*) and its conifer-inhabiting allies have straw-colored or reddish berries.

Effect upon the Host.—The effect of mistletoe upon its host is variable, and may be very slight or pronounced on a given species, while some hosts are more noticeably disturbed. Based on the studies of mistletoe in the Southwest, some of the reported effects are: (1) more or less hypertrophy of the branch at the point of infection; (2) atrophy and final death of a branch beyond a mistletoe tuft, so that the mistletoe comes to occupy the end of a branch, as is frequently well illustrated in the water oak (*Quercus nigra* L.); (3) deforming of branches or even of the main trunk due to infections of long standing; (4) the excessive formation of shoots by the host, giving rise to a sort of witches'-broom effect, as in many infections on the osage orange; (5) delay of the host in putting out its leaves in the spring, noted frequently in cases of heavy infestation; (6) retardation of growth with its stunting and dwarfing effect. York (1909) has expressed doubt as to whether mistletoe ever kills its host, while Bray (1910) states that "mistletoe not only causes mechanical injury, but saps the vitality of the branch and when sufficiently abundant often the whole tree; and in case of the hackberry especially, often results in the death of the entire tree." It has been claimed that the European mistletoe (*Viscum album*) is nourished by its host in the summer, while it, in turn, furnishes its host with food during the winter, but for our American mistletoes no such harmonious relations are believed to exist, the parasite always constituting a drain upon the host.

Etiological Relations.—The fleshy, viscid berries are to some extent an object of food for certain birds, and the seeds are disseminated very largely by these birds, for the most part "by being wiped from the beak against a branch in the bird's efforts to remove the adhesive pulp" or also through the excrement. "In either case the pulp still remaining about the seed causes it to stick to the branch and by drying to become firmly pasted there" (Bray, 1910). The sticky character of the berry pulp is so pronounced that mistletoe fruits in Europe are used in the preparation of bird lime. Mocking birds, cedar birds or wax wings, cardinals, robins and sparrows are reported to be important agents of dissemination, the

first being named the chief distributor. The mocking birds seem to give first choice to other sources of food—for example, hackberry fruits—but when these become exhausted turn their attention to mistletoe berries. Later in the spring when the mistletoe berries soften, they may be broken from their attachment by winds or rains, and fall to other branches on which they become crushed.

By February or later the seeds germinate, the exact time, of course, depending upon the temperature and moisture. The radicle turns towards the substratum and when it comes in contact with it enlarges and becomes flattened to form a more or less circular *attachment disk*.

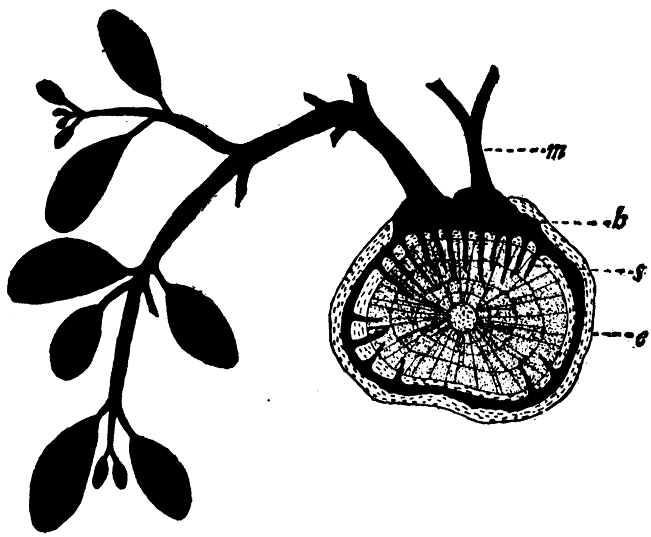

FIG. 251.—Cross-section of a branch infested with mistletoe. *M*, shoot of mistletoe; *h*, haustorium; *s*, sinker; *c*, cortex of host.

A papilla-like projection, the *primary haustorium*, develops from the under surface of the attachment disk, which by mechanical force and the secretion of a digestive substance sinks itself into the bark of the host . . . As soon as the primary haustorium becomes established, it spreads out in the cork cambium, but does not penetrate beyond it (York, 1909).

Sinkers are generally formed during the first year of growth and reach the wood, while buds of the first shoots are formed on the attachment disk. The rate of growth is very slow and by the end of the second year the shoot has produced its first pair of foliage leaves. Mature plants of the Texas mistletoe (*P. engelmanni*) may reach a length of 1 to 3 feet, and in specimens of maximum size have been estimated to be at least 20 years old. With continued growth the cortical haustorium may completely encircle a branch and new shoots may appear even on the side opposite the original point of entrance. There appears to be no fixed limit to the continued existence of mistletoe on its host. The European mistletoe

has been found with sinkers extending through 60 to 70 annual rings of wood, while it is the belief that many infections of the Texas mistletoe are nearly as old as the host itself. According to observations made previous to 1910, there were many "cases in Texas where mistletoe has been repeatedly broken from large branches during the past 20 or 25 years" (Bray, 1910).

Physiological Strains.—The European mistletoe (*V. album*), according to Von Tubeuf (1923), shows three different strains or races: (1) the pine mistletoe (Kiefermistel) on pines and some other hosts; (2) the fir mistletoe (Tannenmistel) on Abies species; and (3) the mistletoe of broad-leaved trees (Laubholzmistel). The American mistletoes have not been studied so intensively and thoroughly as *V. album*, but it is stated that *P. flavescens*, the eastern mistletoe, most generally affects only one of its numerous hosts in a given region, "doubtless illustrating the same host adaptation as the mistletoe of northern Europe" (Trelease, 1916). Bray records 32 different hosts for the Texas mistletoe (1910) and states that it seems likely "that the central Texas form of mistletoe may be more or less freely established upon all of the hosts by seed carried from the mistletoe growing upon any one of them." The marked similarity of several of the recognized species, whose separation has been based largely on the study of herbarium specimens, suggests that more detailed field studies and cultures might be undertaken with profit. Recent studies (Harris *et al.*, 1930) have shown that the Loranthaceæ in general show a higher osmotic concentration of the cell sap than their hosts, but an exception to this rule has been noted in the desert mistletoe on the creosote bush.

Control.—The problem of mistletoe control in America is concerned primarily with shade trees and in a few environments with fruit trees. Two entirely different demands are to be met: (1) the prevention of the development of serious infestations on developing trees; and (2) the extermination of the mistletoe or the mitigation of the damage in heavy infestations of long standing. If trees in mistletoe regions are carefully watched, it should not be difficult to hold the parasite in check by ordinary pruning operations, since infected branches may be cut off a few inches below the point of infection, and the parasite effectually removed. In established infestations which have been permitted to develop unmolested for some years, two courses are open: (1) the breaking of the mistletoe, which retards its growth and gives some relief to the tree; and (2) cutting out of the mistletoe, with treatment of the haustoria to prevent the reappearance of new shoots, combined with heavy pruning or heading back of the host in accordance with its landscape relations or tolerance to severe mutilation. The breaking of mistletoe can be accomplished in tall shade trees by means of a mistletoe hook, consisting of a curved or L-shaped iron inserted in the end of a long pole. With this

implement the rather brittle mistletoe tufts may be pulled or broken from their point of attachment. In this treatment, however, the haustoria give rise to new aerial shoots, which continue the growth of the parasite.

Mistletoe, however, can be kept well under control by cutting off these successive crops of sprouts, and where this is done every year or two the trees are kept more sightly and the damaging effects of the mistletoe reduced to a negligible quantity (Bray, 1910).

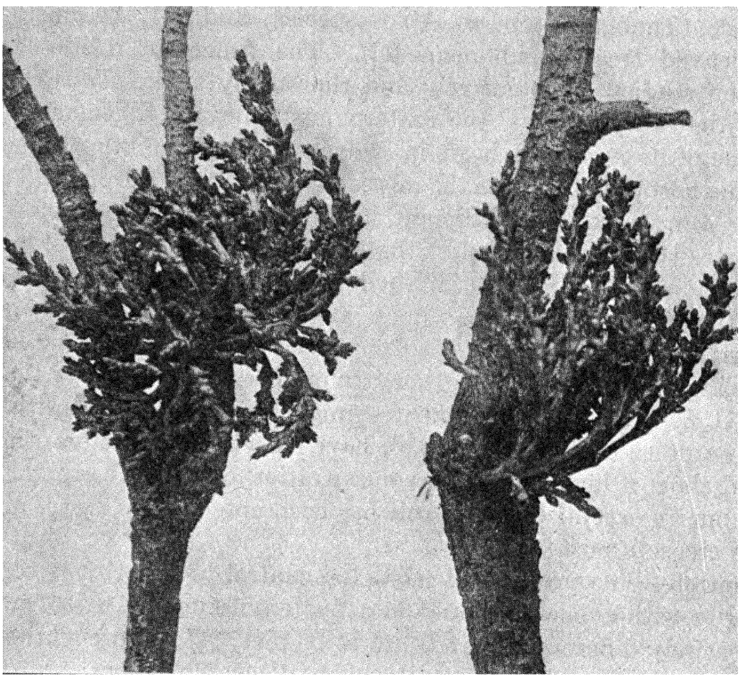

FIG. 252.—Scaly mistletoe (*Razoumofskya sp.*) on pine.

There are conflicting reports as to the success of painting infected regions, after breaking or cutting off of the mistletoe, with various creosote preparations, but it seems that this treatment offers no sure remedy. Apparently, a complete killing of the mistletoe can only be accomplished when the bark covering the haustorium is cut away together with the external parts of the haustorium and the cut surface treated with creosote or coal tar or a combination of the two. This treatment is feasible and successful in infections of moderate size, but in old infections with widely ramifying haustoria is difficult, and much less likely to be successful.

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CHAPTER XXVIII

NEMATODES AND THE DISEASES THEY CAUSE

NEMATODES

Nematodes live free either in moist earth or in water, in decaying vegetable or other organic substances or as ecto- or endoparasites on plants or animals. They feed mostly on juices which they suck up directly from the organic materials of the substratum or which they make available from living cells by boring into them with a buccal spear. Free-living forms which live in the soil and feed upon dead or diseased plant parts may influence plant growth, but the true parasites are of the most phytopathological interest.

General Characters of Nematodes.—The nematodes, or nemas, sometimes called roundworms, have tubular or filiform bodies, with mouth and well-developed alimentary canal. The mouth is provided with papillæ or lips or with hooks or spines in the oral or buccal cavity, and leads into a narrow œsophagus, which usually has thick muscular walls and a cuticularized lining and may be dilated into one or more muscular œsophageal bulbs or pharynx. The œsophagus is a suctorial tube, which by valves and its muscular walls pumps in fluid food and in some species solid particles, and passes it on to the intestine by peristaltic action. The intestine is usually a straight tube which continues the alimentary canal into the rectum, which opens by the anus near the posterior end of the body on the ventral surface. As a result of degeneration the external opening or even the entire alimentary canal may be wanting in certain genera.

The body wall is muscular, and encloses a body cavity containing the blood fluid, the alimentary canal and the excretory and reproductive organs. There is no definite circulatory system, and respiration organs are lacking. The body is unsegmented, but the stiff cuticle is often transversely ringed. The muscular body wall makes it possible for the body to be knotted, curved or bent, and permits the characteristic undulatory movements of filiform species. In most forms two lateral regions remain free from muscle cells and form the so-called *lateral chords or lines.* Dorsal and ventral median chords may also be distinguished.

The sexes are generally separate and the males smaller than the females. The females lay eggs but in a few cases they may bear living young. Many species are parasitic during either all or a part of their life cycle, and some develop in two entirely unrelated hosts. Animal

parasites live mostly in the intestine or other organs of various animals including man, domestic animals and other mammals, insects, etc. Notable forms are *Trichinella spiralis* (Trichina), the cause of trichinosis in man, a disease which is contracted by eating uncooked pork in which the worms are encysted; the Guinea worm (*Filaria medinensis*), which develops in the subcutaneous cellular tissue of man in the tropics of the Old World, and forms abscesses when the worms reach full size (2 feet or more long); and the hookworm (*Necator americanus*), the cause of the hookworm disease, now known to be a terrible scourge in the southern United States, where it is common among the negroes and poor whites. Hookworms live in the soil and gain access to the human body through the soles of the feet, causing "ground itch" or "foot itch," and later pass into the intestines. Hookworms cause anemia, hemorrhage, heart weakness, emaciation and perverted taste, as shown by clay or dirt eating.

Classification of Important Plant Nemas.—Of the various families of Nematoda, the plant pathologist is especially interested in one, the *Tylenchidæ*. This is a family of minute forms, which live free in the soil, water, etc., or on or in plants as parasites. The alimentary canal has two pharyngeal or œsophageal bulbs, the head end is blunt, with central mouth, the posterior end generally pointed. The males have two similar *spiculæ*. The following genera are the most important ones furnishing plant parasites:

Tylenchus.—Buccal spear short, three lobed at the base; œsophagus indistinct with strong, oval, anterior œsophageal bulb and posterior œsophageal swelling joining the intestine by a broad base; male with single testis, and smooth bursa.

Important species: (1) *T. dipsaci* (Kühn) Bastian, the stem nematode of clover, alfalfa, strawberry, hyacinths, etc.; (2) *T. tritici* (S.) Bastian, the cereal and grass nematode; (3) *T. pratensis* De Man., a species infesting cotton and potatoes (Cobb, 1917); (4) *T. similis* Cobb, parasitic on roots of banana and sugar cane (Cobb, 1915).

Tylenchulus.—Spear and œsophagus typically tylenchoid; ectoparasites.

Important species: *T. semipenetrans* Cobb, the root nematode of citrus.

Heterodera.—Buccal spear tylenchoid; male with one testis and without bursa; passes through a "metamorphosis"; female when sexually mature, enlarged flask-shaped, cyst-like and filled with eggs or embryo nematodes.

Important species: *H. schachtii* Schmidt, the sugar-beet nematode.

Caconema.—"Resembling Heterodera, but truly endoparasitic and less specialized in its parasitism; having the amphids protected by 'cheeks'; the males with two testes" (Cobb, 1924). Recently separated from Heterodera with *C. radicicola* as the type.

Important species: *C. radicicola* (Greef) Cobb, the cause of root gall or root knot in hundreds of species of plants.

Aphelenchus.—Buccal spear tylenchoid; male without bursa, direct development.

Important species: (1) *Aphelenchus olesistus* Ritz. Bos., the leaf nematode of ferns, begonias and other greenhouse plants; (2) *A. fragariæ* Ritz. Bos., the cause of the "cauliflower" disease of strawberries; (3) *A. cocophilus* Cobb, the cause of the "red ring" or "rot disease" of coconut; (4) *A. ritzema-bosi* Schwartz, the chrysanthemum-leaf nematode (Steiner, 1924).

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NEMATODE DISEASE OF WHEAT

Tylenchus tritici (S.) Bast.

This is an eelworm disease which affects the aerial parts of the host, but invades especially the inflorescence, transforming the kernels into galls, which resemble the smut balls of stinking smut. Various names, such as "smutted," "bunted," "hard smut," "cockle," "bin burnt" and "immature wheat," have been applied by farmers and mill men in this country. In England it is commonly called "purples," because of the color of the galls and also "false ergot;" in France, "blé nielle," because of the similarity to bunted wheat; and in Germany "Radekrankheit," because at first associated with the seed of cockle (*Agrostemma githago*),

a common weed of wheat fields, also "Gichtkrankheit" on account of the gnarled appearance of infected plants. This disease is distinct from the bulb disease (*T. dipsaci* (Kühn) Bastian) which attacks wheat, rye and oats in addition to numerous other hosts.

History and Geographic Distribution.—This disease was first noted in England by Needham in 1743, but the real significance of the accompanying nematodes was not understood until some years later when Roffredi (1775–1776) showed their causal relation to the malady. The pathogene was first named *Vibrio tritici* by Steinbuch in 1799, and various other names were employed, previous to the appearance of the classical monograph (1865) by Bastian, in which it was transferred to Tylenchus. Of the numerous contributions to our knowledge of the disease which appeared following Roffredi's publication, two are of outstanding importance: (1) The monograph by Davaine (1857); and (2) the contribution of Marcinowski (1909), which added to our knowledge of the pathogenicity and physiology of the parasite. The former "gives practically all that is known today about the etiology of the disease, lucidly describes and illustrates the different stages in the development of the parasite, and records the results of physiological researches on the nematode which furnish a basis for its control" (Byars, 1920).

The disease was first recorded in the United States by Johnson (1909) from collections made in California, but was not found again until 1917 when Fromme reported it from Virginia. It has since been reported from a few other states (West Virginia 11 counties, North Carolina two counties and South Carolina and Georgia one county each), but it has been most prevalent in Virginia (53 counties). The disease has been considered in some detail by Byars (1918, 1919, 1920), Fromme (1919), Coleman and Regan (1918) and Leukel, 1924.

This nematode disease has almost a world-wide distribution, having long been prevalent in the various European countries. It has been noted in Australia and South America and in 1917 Byars reported its occurrence in China, but it seems to be absent from Africa. The disease is believed to be endemic in Europe, and has probably been spread from England or the Continent with exportations of seed wheat. It is fortunate that the disease has not yet been introduced into the principal wheat-growing sections of the United States.

Symptoms and Effects.—The disease may be noted in young plants by the wrinkling, rolling and distortion of the leaves and by the enlargement of the stems of affected shoots. This condition may be noted on young plants in the fall or in the early spring before heading. Small, raised, rounded areas may appear on the upper surface of mildly infected leaves and these lose their normal green color, become yellow, wilt and die. Occasionally very young leaves may "contain light-colored swellings or galls, one or more of which may be located along the midrib, on the leaf edge or between the two, and are misshapen by an unequal lateral development" (Byars, 1920).

When seedlings become more severely attacked,

. . . the young leaves become so strongly infected within the older leaf sheaths that instead of growing straight up normally they may be forced through the latter, carrying along with them the young stem. In this way stems are sometimes bent, and induced to grow in an almost horizontal direction. The leaves become so wrinkled, twisted and rolled as to lose all semblance of their natural

shape. Their normal green color then disappears and finally, after wilting, the entire plant dies (Byars, 1920).

Leaf and stem symptoms are not so noticeable in older plants, but these may be dwarfed, yellowish and exhibit some curling of the upper leaves, or these symptoms may be entirely absent.

The effects of the disease are especially evident in the heads, which are generally shorter, have more widely divergent glumes, and are greener and remain green longer than normal heads, thus resembling some of the

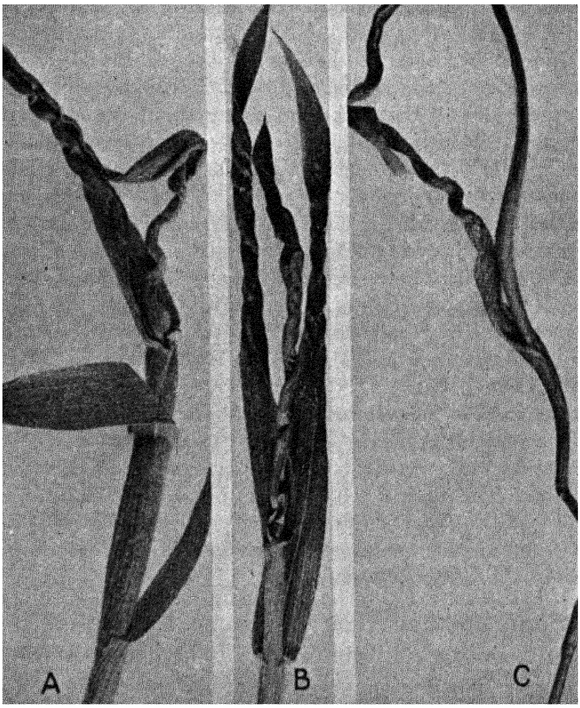

FIG. 253.—Malformation of wheat leaves and stems due to the nematode (*Tylenchus tritici*). (*After Leukel, Jour. Agr. Res.* **27**, 1924.)

effects of stinking smut. All or a part of the flowering glumes of an affected head may contain hard, light-brown to dark-colored galls, which have replaced the normal grains. These galls are smaller than normal wheat grains, but sometimes thicker, and have a much less evident suture or longitudinal furrow. Since they may be mistaken for bin-burnt grains, smut balls or cockle seeds the distinctive characters of each may be noted: (1) *nematode galls* hard, difficult to break, surface smooth, without brush at the tip; wall thick, enclosing a central yellowish-white mass; (2) smut balls fragile, easily broken, surface smooth, brush generally evident; wall thin, enclosing a black powdery mass; (3) cockle seed

more rounded, black, surface covered with rows of short spines; (4) bin-burned grains same as normal grains in form, but discolored.

The injury from the nematode disease may be due to (1) the killing of seedlings, thus reducing the stand; (2) to weakened vitality and lessened yield and a lowering of the market grade or quality of the crop. The injury from the disease is exceedingly variable, as may be noted from the fact that few seedlings may be killed, while in extreme cases a killing of 90 per cent has been recorded. In Virginia, samples of grain from millers and farmers showed 0.1 to 25 per cent of galls, and in one case they exceeded 50 per cent. In considering the significance of the

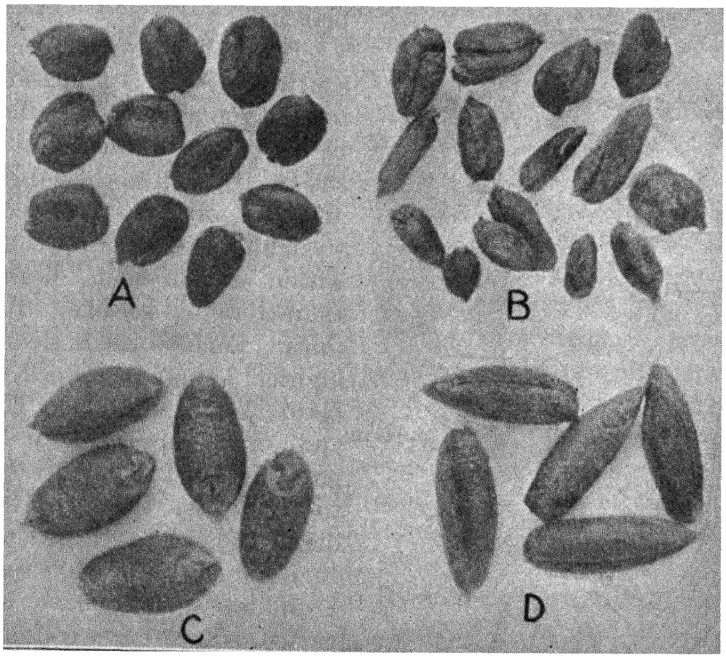

FIG. 254.—Nematode galls and normal grains. *A*, galls from wheat; *B*, galls from rye; *C*, normal wheat kernels; *D*, normal rye kernels. (*After Leukel, Jour. Agr. Res.* 27, 1924.)

disease in these cases it should be borne in mind that many galls are lost before and during threshing, and that in severe infections there is much injury that is not reflected in the nature of the threshed product. The quality or market grade is lowered because of the reduced test weight per bushel, and the increased amount of dockage, while the "flour yield from such infected wheat is reduced, and the percentage of low-grade flour and shorts is increased" (Coleman and Regan, 1918). The galls are difficult to remove by screening or fanning, tests showing 88 per cent after using the oat-kicking machine, 65 to 70 after using the three standard sieves and 40 to 45 per cent after vigorous fanning with 850 revolu-

tions per minute. The galls are lighter than sound wheat, with a specific gravity of 0.8125, so they can be removed by floating. Use can be made of wheat washers and driers that are especially devised to handle smutty wheat.

Etiology.—This disease is due to *Tylenchus tritici* (S.) Bast., one of the parasitic nematodes, belonging to the family Tylenchidæ. The pathogene may be found in the galls on young leaves, in the flower galls, as an ectoparasite within the leaf sheaths or it may exist for a time in the soil. The parasitism of this organism has repeatedly been demonstrated by inoculations made by planting whole or broken galls with wheat or by pouring an infusion of galls over the seed or into the trenches before planting.

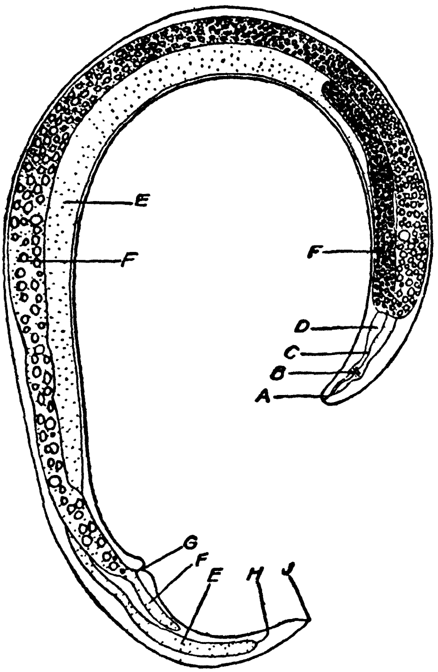

FIG. 255.—Lateral view of young female of *Tylenchus tritici* (for explanation of letters see Fig. 256). (*After Byars, U. S. Dept. Agr., Bul.* 842, 1920.)

The yellowish-white center of a nematode gall is a mass of these minute roundworms in what may be designated as their *second larval stage*. If a gall is soaked in water and then opened, the freed larvæ straighten out, and later, sometimes within a half hour, begin their characteristic, eel-like, vital motions. Each worm is slender, cylindrical to spindle shaped, slightly blunt at the anterior or head end and tapers to a point at the posterior end. They are from 658 to 910μ in length and 15 to 20μ in diameter. The average length has been given as 869μ, while specimens 1 millimeter long have been reported. Each larva consists of an outer tube or body covering enclosing a smaller tube, the alimentary canal, the space between the two being the body cavity. The mouth opens into a buccal cavity containing a buccal spear, 9 to 11μ long, which is hollow, pointed at the free end and trilobed at the base.

By means of this hollow spear, which can be exserted and retracted by strong muscles, the larva is able to pierce its egg membranes, to force its way into plant tissues and by means of the fine canal which extends through the spear to absorb plant juices or other liquid foods.

Below the spear and buccal cavity the digestive canal is continued as a slender œsophagus, which shows an anterior, globular, œsophageal bulb, or muscular enlargement. The slender digestive canal is continued

back of this bulb and gradually enlarges into a second or posterior, elongated œsophageal bulb, which joins the much larger intestine, opening first into a short narrow rectum leading to the anus or vent about 50μ from the point of the tail. Near the center of the anterior bulb there is "a small valve capable of expansion and contraction by the muscular wall of the bulb," which by a pump-like action sucks liquids through the spear, and passes them on into the intestine. The intestine contains translucent granular matter and refractive nuclei of its wall cells can also be plainly seen, while a half-moon-shaped, light zone, containing the primordia of the reproductive organs, may be noted at about the middle of the body. At this stage there is no sexual differentiation.

The life history of the pathogene may be briefly outlined. The galls falling to the ground or mingled with the seeded grain decay and the larvæ escape into the surrounding soil. By their own activity they are able to migrate through the soil for a radius of 20 to 30 centimeters. They may live free in the soil for several months, but if they find no host plant to infect they finally perish. When they come in contact with susceptible seedlings, they penetrate between the leaf sheaths near the apical or growing points of the culms, and remain in this position until the head is produced, or give rise to the curled and rolled leaves and leaf galls, as described under Symptoms. When the heads are formed some of the larvæ that are lying in wait enter the flowering parts, presumably by the piercing action of their buccal spears, and within the galls formed from ovaries or adjacent organs become sexually mature, pair and lay thousands of eggs. The old worms die, the eggs hatch and the newly formed larvæ become dormant by the time the galls have reached maturity. Sexual maturity may also be attained in some of the leaf galls. A leaf gall may contain a few to as many as 25 worms, while two to 25 or more may infest a single flower.

The sexually mature males and females develop within the galls, and show modifications in structure and size. Females are 3.5 to 4 millimeters in length and 168μ or more in diameter. The most of the body cavity back of the œsophagus is occupied by the egg-producing organ, which opens at the vulva some distance in front of the anus. The egg-producing organ consists of a short posterior sterile branch and a large anterior fertile portion which is so long that it is folded at the front and again near the middle. The first portion of the fertile branch is a glandular, tubular portion, the *uterus,* which expands into a vesicular-shaped portion directly connected with the ovary. The uterus may be filled with fertilized eggs in various stages of development. A single female may lay more than 2000 eggs and thus from the adults of a single gall 10,000 to 90,000 larvæ may result.

The males are smaller than the females, 2 to 2.5 millimeters long and more slender. In front of the pointed tail end there is a curved trans-

parent wing, the bursa, near whose center the intestine and reproductive organ open. The latter extends as a tube of about uniform diameter nearly to the œsophagus, where it folds back for a short distance. The testis tube may be filled with mature or developing spermatozoa. Special copulatory organs, the spiculæ, are located near the sexual opening.

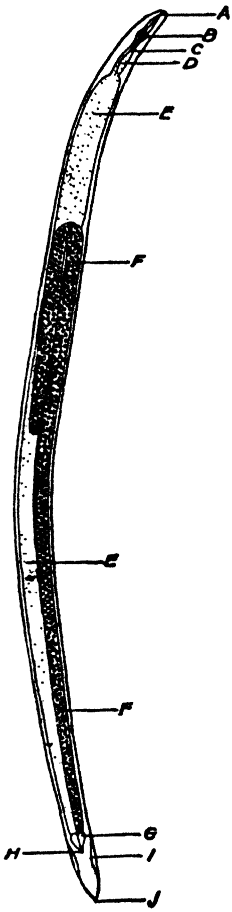

FIG. 256.—Ventral view of young male of *Tylenchus tritici*. *A*, spear; *B*, anterior œsophageal bulb; *C*, œsophageal canal; *D*, posterior œsophageal bulb; *E*, digestive system; *F*, reproductive system; *G*, spicula in the male and vulva in the female; *H*, anus; *I*, bursa of male; *J*, tail. (*After Byars, U. S. Dept. Agr. Bul.* **842**, 1920.)

The eggs are elongated ellipsoid, filled with dense rounded granules, possess a single central light spot, the nucleus, are covered with a tough, transparent skin and measure 73 to 140 by 33 to 63μ. Segmentation may be begun before oviposition, but is generally delayed until the eggs are laid. It proceeds rapidly and within a short time the young larva pierces the egg membrane with its buccal spear and escapes from the shell. The larvæ pass into a dormant and very resistant state by the time the galls are mature. In this condition they are able to remain dormant for many years and resume their life cycle when favorable conditions of moisture are offered. Byars reports the successful reactivation of larvæ (1920) from galls in wheat imported from Turkestan in 1910, and cites a case of resumed vital activity after a dormancy of 27 years.

The dormant larvæ in the galls are very resistant to temperature changes or to the action of chemical agents. Galls soaked several days in tap water and then immersed in hot water gave the following results: 46°C. for 5, 10 or 15 minutes, no effect; 48°C., ditto; 50°C. ditto; 50°C. for 30 minutes, all killed; 52°C. for 20 minutes, all lifeless; 54°C. for 10 to 15 minutes, practically all killed; 54°C. for more than 15 minutes, all killed; 56, 58 or 60° for 5 minutes, all killed. The larvæ in dry galls are much more resistant than when moist. They are able to stand temperatures of 58°C. or less for 5 to 20 minutes, with only pronounced reductions in the per cent alive, while it required 60°C. for 10 minutes to kill all of the larvæ. Larvæ outside the galls in water are more sensitive to heat than when in the galls, being killed by half the length of exposure necessary for whole galls. The larvæ in moist galls will withstand soaking in formaldehyde solutions that are sufficiently strong to cause more seed injury than can be tolerated.

In fall seedings, the method of overwintering of the pathogene has been disputed, some investigators contending that the larvæ overwinter in the galls and escape into the soil and infect the young plants in the spring, while others believe that they are set free and live in the soil or locate in the seedlings. Most of the evidence favors the fall infection, but it seems that the exact behavior may depend on temperatures, time of seeding and available moisture. Apparently, ordinary winter temperatures would not be fatal to free-living nematodes of this species, as they have been subjected to 15 to 18°C. below zero without injury.

> Only in its second larval stage does the parasite constitute a source of infection. All other stages of the life cycle are more or less transitory, unable to withstand unfavorable conditions, and unable to live or develop for any appreciable time outside the host plant (Byars, 1920).

Because of this behavior, the galls mingled with seed wheat must be the principal agent by which the disease is introduced into the field or spread to new localities. Free larvæ, mingled with the soil, may remain alive sufficiently long to be transported long distances, but it is believed that this method of dissemination is of very minor consequence.

An interesting relation between the so-called Dilophospora disease (*Dilophospora alopecuri* (Fr.) Fr.) of cereals has been established by Atanasoff (1925). This disease had been supposed to be caused by the accompanying fungus, but Atanasoff claimed that the reputed pathogene can affect only hosts that are attacked by *Tylenchus tritici*. The nematodes carry the spores of Dilophospora to the growing points and affect the host in such a way that the fungus is able to develop. This is not, however, in agreement with later studies (Schaffnit and Wieben, 1928) in which successful inoculations were made with the fungus without the intervention of the eelworms. It seems probable that the eelworms do act as carriers and also increase the severity of the fungous infection. A bacterial disease attacking only plants infected by eelworm has also been described (Carne, 1926). This produces a yellow slime in the heads and adjacent leaves.

Host Relations.—*T. tritici* is primarily a parasite of wheat, but it is also able to infect some other cereal hosts. It has been noted under natural conditions on spelt, and by artificial inoculations an abundant infection of emmer, rye and spelt, and faint infections of oats and barley have been reported. Some inoculations on barley have yielded no galls, and on oats only a very few minute flower galls, hence barley and oats appear to be very resistant, while rye, emmer and spelt are quite susceptible. Flower galls due to nematodes similar to or identical with *T. tritici* have been studied on various wild grasses (Bessey, 1905) in both Europe and America. Investigators have mostly agreed that the slight morphological differences shown by the different collections are not suffi-

cient to justify their retention as distinct species. Attempted inoculations and observations of wild grasses growing with infected wheat indicate that the various flower-infesting nematodes of wild grasses are either biological strains of a single morphological species or represent distinct species, since none of the grass forms have been communicated to wheat, and the wheat nematode has not given successful infections on any of the wild grasses.

No extensive tests of varietal susceptibility are available, but Fromme (1919) records his experience with five varieties which showed from 44 per cent of infected heads in Red Wonder to 74 per cent in Fultz, while Fulcaster showed the highest per cent of galls in the harvested grain (31 per cent) and Leaps' Prolific the lowest (23 per cent). Kanred is reported as very resistant (Leukel, 1929), but this variety is not adapted to the infested regions.

Control.—In considering the prevention of nematode infestation of wheat the following facts should be kept in mind: (1) The galls in seed grain constitute the principal source of infection; (2) galls that fall to the ground during harvesting operations of an infected crop may contaminate the soil; (3) wheat is the principal crop that suffers from the disease, but other cereals may develop light infections; (4) the wheat nematode can survive only in connection with its host, either in the galls or on a living host. With these facts in mind, the use *of clean or nematode-free seed wheat and the rotation of crops* are suggested.

Clean seed may be obtained in either of several ways: (1) by the selection of seed from localities known to be free from the disease, either in the home environment or by sending away if necessary; (2) by the separation of the galls from contaminated seed if it must be used; and (3) by the hot-water treatment to kill the nematodes carried by the galls. Treatment of seed is advised only when it is impossible to obtain clean seed or when it is desirable to retain a valuable variety.

Neither sieves nor fanning mill will separate all the galls but a complete separation may be effected by the salt-brine treatment. Pour the contaminated seed into a 20 per cent solution of common salt (40 pounds to 25 gallons) and stir vigorously to bring the light seed, galls or other trash to the surface. Decant the floating material into a second container covered with a cheese-cloth screen, then rinse the clean wheat in pure water and spread out to dry. The skimmings should either be burned, or if it is desirable to use them for stock feed, they may be soaked in boiling water to destroy the nematodes. As an extra safeguard to kill any nematodes that may be on the surface of the grain, the cleaned seed may be treated with hot water for 10 minutes at a temperature of 51 to 52°C.

Equally good results may be obtained by the hot-water treatment alone. By this method the grain should be soaked for 1 hour in unheated

water and then immediately immersed in hot water, using any one of the following effective combinations: 20 minutes at 52°C., 15 minutes at 54°C. or 10 minutes at 56°C.

Crop rotation must be practiced if the previous crop of wheat was infected. Some of the heaviest losses have occurred in land cropped to wheat for a number of years in succession. The consensus of opinion favors growing a non-susceptible crop for three consecutive years, although two may suffice. For the first year a cultivated crop would seem preferable, since it will be desirable to eliminate all volunteer wheat and any other grass species which may possibly act as hosts. It should be noted that rye, emmer, spelt, oats and barley are to be classed as susceptible crops and so should be avoided during the period of the rotations before the return to wheat. Until more specific information is available as to the communicability of the wheat nematode to certain wild grasses, all those which are known to produce nematode flower galls should be avoided. Fromme recommends clover, alfalfa or some other legume for one or two years to be followed by corn for one year, before returning to wheat.

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ROOT KNOT OR ROOT GALL

Caconema radicicola (Greef) Cobb (*Heterodera radicicola* (Greef) Müller)

This disease is characterized on most of its hosts by the formation of characteristic enlargements on the roots, which have suggested other common names than root knot, such as "root gall," "eelworm disease" and "big root."

History and Geographic Distribution.—The root knot was first discovered by Berkeley in 1855, but it was not until 1872 that the nematode was studied from galls on *Dodartia orientalis* by Greef and named *Anguillula radicicola.* The first exhaustive study of the parasite was made by Müller in 1883 and presented as an inaugural dissertation. The biological and phytopathological aspects of the disease were well treated by Frank in 1885. Monographic treatments followed in 1889 by Atkinson, in 1898 by Stone and Smith and in 1911 by Bessey. Much of the more recent literature has dealt with the various methods of control, only a few workers giving consideration to ecological and etiological aspects (Godfrey, 1924, 1926).

The root-knot nematode is supposed to be a native of the tropical and semitropical regions of the Old World, whence it has spread to all countries of the world. Its range in the open is limited only by its temperature requirements, which confine it mainly to the warmer regions, but under the favorable conditions of greenhouse culture in any part of the world it frequently becomes a very destructive pest. Root knot is common and often very serious on field-grown crops in the South Atlantic and Gulf states and California. Sporadic field occurrences have been reported as far north as New York and Michigan and west of the Rocky Mountains from Idaho, Oregon and Washington. In New Jersey, Maryland, Delaware, Southern Ohio, Indiana and Illinois it is found frequently in truck gardens and nurseries, but it is mainly south of this zone that the disease becomes an outstanding problem in crop production.

Symptoms and Effects.—The manifestations of the disease and its effects are: (1) the galls or enlargements on the root system; (2) dwarfing and retarded growth with more or less root killing and reduction of yield; (3) a paler-green color of the foliage than normal, frequently accompanied by marginal necrosis; (4) a more pronounced wilting in hot, dry weather than for healthy plants; (5) the destruction of seedlings almost as soon as they get above the surface of the soil, very similar to fungous damping-off, especially in heavily infested soils, resulting in thin or irregular stands; and (6) the premature death of older plants or even of those approaching seasonal maturity. The degree of expression of these effects is measured by the severity of infection as indicated by the size and abundance of the root galls.

The root galls or enlargements may appear as small, scattered, tubercle-like growths or as extensive swellings of either large or small roots. The knots may vary in size from scarcely noticeable swellings to hypertrophies an inch or two in diameter and in extreme cases may involve nearly the entire root system (see Fig. 257). The galls on violets are the smallest, and the largest have been recorded on roses (size of duck eggs). The galls on Dicots are most frequently, sharply defined tubercles, while on Monocots they are generally slender spindle-formed enlargements. If the knots are cut across, from one to several dark specks may be noted in the cortex, marking the location of the pathogene. In the Irish potato the tuber suffers more than the roots and when infested

FIG. 257.—Roots of tomato plant showing severe invasion by root-knot nematodes. (*After G. F. Atkinson.*)

may show a rough, warty surface. Somewhat similar characters are to be noted in fleshy roots like carrots or turnips. Such tubers or roots when cut across will show small, brown, necrotic spots just below the skin (Fig. 258), in which enlarged gravid or young larvæ may be found. Affected organs, if carefully dissected or broken open, may show the enlarged female cysts as pearly white, rounded or pyriform bodies, large enough to be seen with the naked eye ($\frac{1}{40}$ to $\frac{1}{25}$ inch).

Etiology.—Root knot is caused by the minute roundworm, *Caconema radicicola* (Greef) Cobb, which penetrates the parenchyma of young roots and by its presence (chemical not mechanical stimulus) induces hyperplasia and also cell enlargement. The pathogene was formerly included in the genus Heterodera but was made the type of a new genus by Cobb.

The young larvæ which enter the tender root tissue in the case of a primary infection develop to sexual maturity within the root, pair and soon the female enlarges and eggs are produced, 300 to 500 in number, extruded in egg masses or oöcysts. The individual eggs are colorless, transparent, oval shaped, about $\frac{1}{250}$ inch long and undergo segmentation either within the body of the mother or after being expelled, and the young larva emerges from the egg membrane through a hole which it pierces. Under conditions of high temperature the egg masses may burst through the side of the root and appear as yellowish, semitransparent bodies closely attached to the root, but at lower temperatures the egg masses remain internal. The young larvæ may migrate in the roots with the production of secondary infections or they may escape into the soil where they may survive for months without any parasitic relation. In the warmer climates the root-knot nematode may pass through as many as 10 to 12 generations in a year, a life cycle from egg to egg being completed in 3 weeks to 2 months.

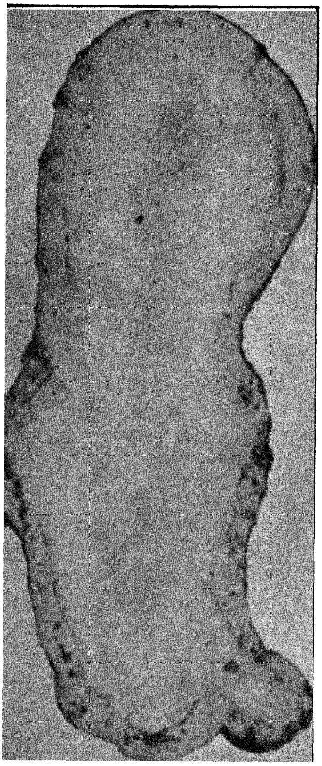

FIG. 258.—Section through a potato invaded by root-knot nematodes. The worms are located in the necrotic areas just beneath the surface. (*After W. A. Orton.*)

The root-knot nematode is sensitive to various environmental factors, such as cold or heat, drought or moisture and toxic chemicals. The egg stage is the most resistant to unfavorable environmental conditions, which suggests that the egg is the overwintering stage.

Environmental Relations.—It is generally recognized that climate is one of the limiting factors in the distribution of the root-knot nematode. According to Godfrey (1926), the amount of root knot below a soil temperature of 16°C. (60.8°F.) is much less than when it is only 2 or 3° higher. It is almost eliminated 3° lower yet, and at 10 to 12°C. infections are very rare. Infestations are frequently severe at temperatures up to 29.5°C. (85°F.) and occur to some extent at much higher temperatures. The thermal death point for larvæ is 128°F.; for eggs 137°F. or 10 minutes at 110°F. for larvæ, and 10 minutes at 119°F. for eggs. It is the temperature relation that is largely responsible for the present geographic range of root knot as a serious pest.

Many successful commercial greenhouse men throughout the country, as well as truck growers in the South, make use of the fact of the warmth-requiring proclivities of the root-knot nematode to grow successfully low-temperature-enduring plants such as lettuce and celery, in root-knot infested ground (Godfrey, 1926).

Lettuce may be grown in greenhouses in nematode-infested soil at 45 to 60°F. with little infection, while cucumbers or tomatoes at 70 to 75° would be severely affected.

The amount of moisture seems to play only a small part in root-knot development, so long as the moisture content of the soil is favorable to the growth of

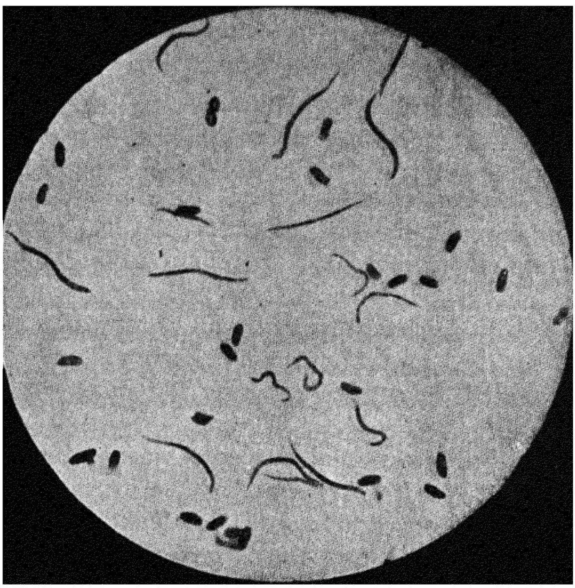

FIG. 259.—Eggs and young root-knot worms just hatched, taken from a potato. (*After F. B. Headley.*)

the crops. Within the range of 40 to 80 per cent of the moisture-holding capacity of the soil there is very little difference in root-knot development. At 60, 70 and 80 per cent there appears to be a slight increase over the other percentages. Even below 40 per cent, which is too dry for good growth of ordinary crops, and above 80, which is more or less muddy, considerable root knot occurs (Godfrey, 1926).

Varying results have been obtained by flooding as a means of killing the root-knot nematodes. Flooding for 3 to 4 weeks failed to give control in a number of cases, but freedom from root knot is reported by Florida truck growers whose lands are flooded for 3 or 4 weeks.

The root-knot nematode is sensitive to drying, the larvæ being more sensitive than the eggs, the former being killed by complete dryness in

3 minutes, and the latter in 2 hours. In the ordinary handling of soils desiccation would not free them from infestation, since nematodes are known to penetrate far below the plow line, and a complete drying would be necessary. Root knot is likely to be most serious on light, sandy or muck soils but less serious in clay soils.

Host Relations.—The root-knot nematode attacks an enormous number of plants, its known hosts including over 500 wild or cultivated species. The following are listed as especially subject to the disease: (1) *field crops*, including alfalfa, clover, cotton, cowpea (exceptions below), sugar beet, sugar cane, sweet potato, tobacco and vetch; (2) *ornamental and drug plants*, represented by begonia, cineraria, clematis, coleus, dahlia, hollyhock, ginseng, goldenseal, peony, rose, sweet pea and violet; (3) *truck crops*, such as asparagus, bean, beet, cantaloupe, carrot, celery, cucumber, dasheen, eggplant, garden pea, lettuce, muskmelon, okra, onion, pepper, potato, salsify, spinach, strawberry, tomato and watermelon; and (4) *woody hosts*, the most important being the almond, catalpa, cherry, date palm, European elm, fig, mulberry, Old World grape vine, peach, pecan, Persian walnut and weeping willow. Many wild plants, including most of the common weeds, are also attacked.

The following list includes the more important cultivated plants which by their immunity or resistance are safe for use in crop rotations on contaminated land: barley, corn, rye, wheat and winter oats; sorghum, milo and kafir corn; red top, timothy and nearly all grasses; Iron, Brabham, Monetta and Victor cowpeas, peanut, Laredo soy bean and velvet bean.

Control.—Two practices stand out as of special importance in the control of root knot: (1) *crop rotation*, or the growing of resistant or immune crops for 2 to 3 years; and (2) *soil sterilization*, the former of first importance in field cultures, the latter for crops under glass or in intensive garden culture. Other practices may be of value in specific cases such as early planting of adapted varieties which are known to mature before the periods of high temperature, the selection of a low-temperature crop for infested greenhouse soils or the cultivation and high fertilization of orchards and ornamental plantings, with the avoidance of susceptible cover crops. Complete fallow with the destruction of all weeds is very helpful, and in some cases benefit has been obtained by flooding for several months. The use of a "catch crop" has been advocated in Germany, that is, growing a susceptible crop and removing the plants and destroying them before the nematodes migrate into the soil.

Under field conditions satisfactory results will follow very careful attention to crop rotation, such as an immune cereal the first year, a very resistant legume the next year to be followed by the desired crop. A suggested rotation for the home vegetable garden which often becomes

very badly infested, owing to continuous cropping to susceptible varieties, is: *first*, year, chicken runs; *second* year, garden; and *third* year, corn, using three plots or chicken runs so that one section is available each year for garden (Godfrey, 1923).

The methods of soil sterilization are: (1) the application of a nemacide or a chemical agent toxic to the nematodes; (2) drenching with boiling water; and (3) steaming. Many different chemical treatments have been tried, mostly with indifferent success. Certain proprietary preparations have proved worthless. One of the most promising was the combined sterilizing and fertilizing treatment with *calcium cyanamide* (Watson, 1917, 1921), but this does not seem to have been generally accepted. *Sodium cyanide* has also been used with some success (Durez, 1917). Drenching with boiling water is adapted to sterilization of pots, flats or small benches and may be accomplished by the use of 7 gallons per cubic foot of soil (Byars and Gilbert, 1920). Steam sterilization is in very general use in greenhouses in regions where nematodes are prevalent and is the most economical and effective control. Using high-pressure steam, various methods of treating the soil are in use, including movable or stationary perforated pipes, the inverted pan or temporary or permanent buried systems of tile (Beinhart, 1918; Brown *et al.*, 1922; Sackett, 1927; Newall, 1930).

Consideration has been given to the possibility of the control of root knot and other nematode diseases by means of predatory nemas (Steiner and Heinly, 1922) and to the selection and breeding of resistant varieties of crops (Malloch, 1923). The recent discovery of two very resistant or nearly immune strains or varieties of pole-snap beans is an illustration of what may be expected by careful selections from large populations grown in heavily infested soils.

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IMPORTANT DISEASES DUE TO NEMATODES

Nematode disease of wheat (*Tylenchus tritici* (S.) Bast.).—(See special treatment, p. 882.)

Stem and bulb nematode. (*Tylenchus dipsaci* (Kühn) Bast.).—This nematode is known to attack over 100 different species of host plants and has recently been reported as the cause of severe injury to strawberries, red clover and alfalfa in the Pacific Northwest. On hyacinths and other bulbs it is known as the "ring disease." GODFREY, G. H.: The eelworm disease; a menace to alfalfa in America. *U. S. Dept. Agr. Circ.* **297**: 1–18. 1923. WILKE, S.: *In* Sorauer's Handbuch der Pflanzenkrankheiten, 4te Auf. **4**: 7–21. 1924. HODSON, W. E. H.: Observations on the biology of *Tylenchus dipsaci* (Kühn) Bast., and on the occurrence of biological strains of the nematode. *Ann. Appl. Biol.* **13**: 219–228. 1926.

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Strawberry nematode (*Aphelenchus fragariæ* Ritz. Bos).—The cause of "cauliflower disease" or dwarf of strawberry. STANILAND, L. N. AND SWARBRICK, T.: Experiments on the relation of strawberry eelworm (*Aphelenchus fragariæ*) to "red plant" and "cauliflower" disease of strawberries. *Jour. Bath. & West & South Co. Soc. Agr.* **6**: 198–209. 1929. GOFFART, H.: *In* Die Aphelenchen der Kulturpflanzen, pp. 33–40. Julius Springer, Berlin. 1930. STEVENS, N. E. and MOOK, P. V.: Field observations on strawberry dwarf in North Carolina. *Phytopath.* **20**: 669–672. 1930.

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